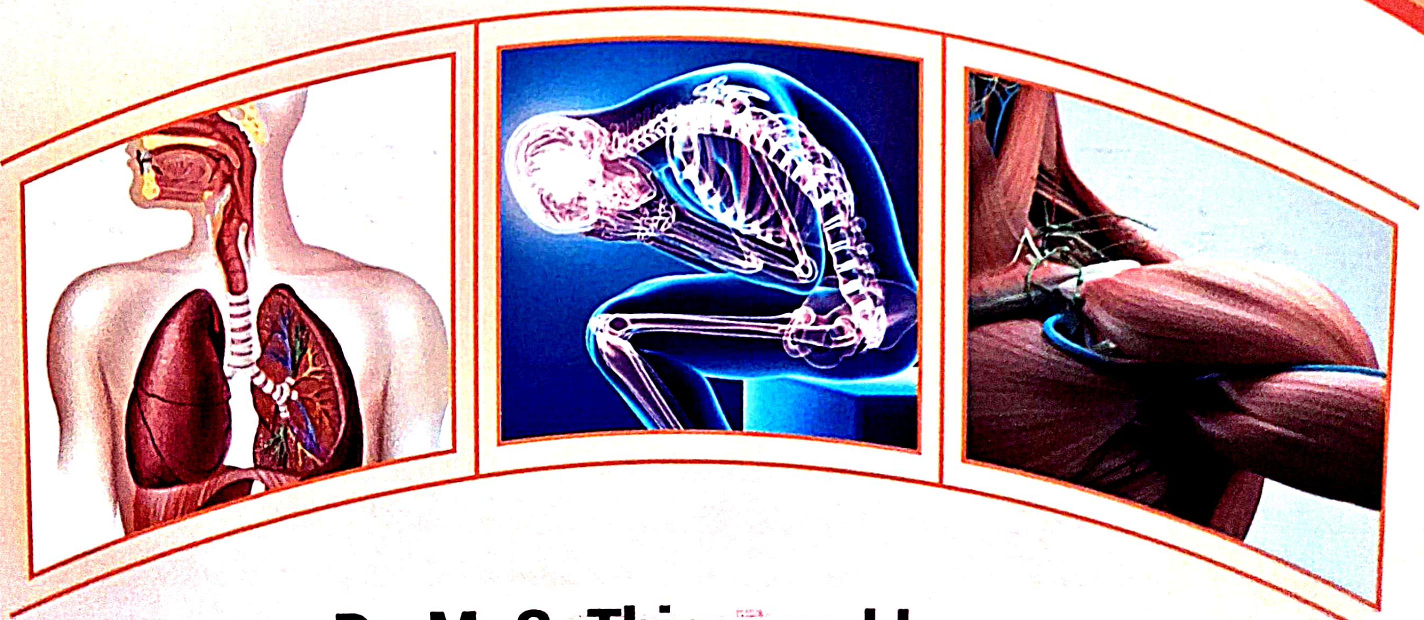
CHAUKHAMBHA VISHVABHARATI
VARANASI

The Anthology of
ROGA NIDĀNA AND VIKṚTI VIJÑĀNA

Aligned with the NCISM Curriculum
Subject Code: AyUG-RN

Volume-2

With chapterwise MCQs, SAQ and LAQ



Dr. M. S. Thirunavukkarasu

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HARIDAS AYURVED SERIES
140

The Anthology of
ROGA NIDĀNA AND VIKṚTI VIJÑĀNA

Aligned with the NCISM Curriculum - Subject Code: AyUG-RN

Volume 2

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Varanasi (Bharat)

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Course curriculum for Second Professional BAMS

(PRESCRIBED BY NCISM)

Roga Nidan evam Vikriti Vigyan

(SUBJECT CODE : AyUG-RN)

Paper -2 : Vyadhi Vigyan, contemporary understanding and updates

Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
Topic 1 **1. Agnimandya – Ajeerna, Anaha, Adhmana, Atopa (Lecture :1 hours, Non lecture: 0 hours)**		
⦿ Define Anaha	MK	11
⦿ Enlist types of Anaha	MK	12
⦿ Describe Adhmana and Atopa	MK	13-15
Topic 2 **2. Chhardi (Lecture :1 hours, Non lecture: 0 hours)**		
⦿ Define Chhardi	DK	19
⦿ Describe hetu and samprapti of Chhardi.	DK	20
⦿ Enlist bheda of Chhardi	DK	21-25
⦿ Describe purvaroopa, samanya lakshana, vishishta lakshana, upadrava and sadhya asadhyata of Chhardi	DK	21
Topic 3 **3. Amlapitta (Lecture :2 hours, Non lecture: 0 hours)**		
⦿ Define Amlapitta	MK	28
⦿ Describe hetu and samprapti of Amlapitta	MK	29
⦿ Enlist bheda of Amlapitta	MK	30
⦿ Describe purvaroopa, samanya lakshana, vishishta lakshana, and sadhya asadhyata of Amlapitta	MK	30-33
Topic 4 **4. Shoola (Lecture :1 hours, Non lecture: 0 hours)**		
⦿ Define Shoola	MK	36
⦿ Enlist Shoola bheda	MK	38
⦿ Describe and differentiate Parinama shoola and Annadrava shoola	MK	44
Topic 5 **5. Atisara, and Pravahika (Lecture :3 hours, Non lecture: 1 hours)**		
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⦿ Describe hetu and samprapti of Atisara	MK	52
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⦿ Describe purvaroopa of Atisara	MK	53
⦿ Enlist and describe upadrava of Atisara	MK	61

Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
⦿ Describe sadhya asadhyata of Atisara	MK	61
⦿ Describe nivrutta Atisara lakshana or vigata Atisara lakshana	MK	62
⦿ Describe samprapti of Pravahika	MK	63
⦿ Enlist bheda of Pravahika	MK	65
⦿ Describe samanya and vishishta lakshana of Pravahika	MK	65
⦿ Differentiate between Atisara and Pravahika	MK	65
⦿ Differentiate Doshaja Atisara	MK	57
⦿ Explain Bhayaja atisara, Shokaja atisara and Raktaja atisara lakshana	MK	59-61
Topic 6 6. Grahani (Lecture :2 hours, Non lecture: 1 hours)		
⦿ Describe pratyatma lakshana of Grahani	MK	71
⦿ Describe hetu and samprapti of Grahani	MK	71
⦿ Enlist bheda of Grahani	MK	72
⦿ Describe purvaroopa, and samanya lakshana of Grahani	MK	72
⦿ Explain Sangraha grahani and Ghati yantra grahani	MK	75-76
⦿ Explain sadhya asadhyata of Grahani	MK	76
⦿ Differentiate Atisara and Grahani Roga	MK	77
⦿ Differentiate Grahani dosha and Grahani roga	MK	76
⦿ Differentiate Doshaja Grahani	MK	75
Topic 7 7. Visuchika, Alasaka, Vilambika (Lecture :1 hours, Non lecture: 0 hours)		
⦿ Define Visuchika	MK	79
⦿ Describe lakshana, upadrava, and sadhyasadhyata of Visuchika ,	MK	80-81
⦿ Alasaka and Vilambika		82-85
Topic 8 8. Common GIT diseases (Lecture :1 hours, Non lecture: 4 hours)		
⦿ Describe the clinical features of Ulcerative dyspepsia and Non-ulcerative dyspepsia, Irritable Bowel Syndrome, and Inflammatory Bowel Diseases	DK	90-97
⦿ Perform relevant clinical examination of Ulcerative dyspepsia and Non-ulcerative dyspepsia, Irritable Bowel Syndrome, and Inflammatory Bowel Diseases	DK	90-97
⦿ Order and interpret relevant investigations of Ulcerative dyspepsia and Non-ulcerative dyspepsia, Irritable Bowel Syndrome, and Inflammatory Bowel Diseases	DK	90-97
Topic 9 9. Mutrakrichhra (Lecture :2 hours, Non lecture: 1 hours)		
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⦿ Describe hetu and samprapti of Mutrakrichhra	MK	101

Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
⦿ Enlist bheda of Mutrakrichhra	MK	102
⦿ Describe vishishta lakshana of Mutrakrichhra	MK	103
Topic 10 **10. Mutraghata (Lecture :3 hours, Non lecture: 1 hours)**		
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⦿ Enlist conditions of Mutra shoshana and Mutra pratihanyate among different types of Mutraghata	MK	110
⦿ Describe different types of Mutraghata	MK	111
⦿ Differentiate Mutrakrichra and Mutraghata	MK	117
Topic 11 **11. Common Urinary diseases (Lecture :1 hours, Non lecture: 6 hours)**		
⦿ Describe the clinical features of Urinary Tract Infection, Prostatomegaly, Nephrotic syndrome, Nephritic syndrome, Acute Kidney Injury and Chronic Kidney Disease	DK	119-130
⦿ Perform relevant clinical examination of Urinary Tract Infection, Prostatomegaly, Nephrotic syndrome, Nephritic syndrome, Acute Kidney Injury and Chronic Kidney Disease	DK	119-130
⦿ Order and interpret relevant investigations of Urinary Tract Infection, Prostatomegaly, Nephrotic syndrome, Nephritic syndrome, Acute Kidney Injury and Chronic Kidney Disease	DK	119-130
Topic 12 **12. Hikka (Lecture :1 hours, Non lecture: 0 hours)**		
⦿ Describe pratyatma lakshana, hetu, and samprapti of Hikka	NK	133-136
⦿ Describe cardinal features of Pancha Hikka	NK	136-139
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⦿ Describe hetu and samanya samprapti of Shwasa	MK	144-145
⦿ Enlist bheda of Shwasa	MK	146
⦿ Describe purvaroopa, and sadhya asadhyata of Shwasa	MK	150
⦿ Describe vishishta lakshana of Tamaka shwasa with its avastha bheda	MK	147
⦿ Describe dosha predominance in Shwasa	MK	146
⦿ Identify cardinal features of Pancha shwasa and correlate with the current science	MK	151
⦿ Differentiate Pancha shwasa	MK	150
Topic 14 **14. Kasa (Lecture :2 hours, Non lecture: 0 hours)**		
⦿ Define Kasa	MK	154
⦿ Describe hetu and samprapti of Kasa	MK	155
⦿ Enlist bheda of Kasa	MK	156

Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
◉ Describe purvaroopa and sadhya asadhyata of Kasa	MK	162
◉ Describe the differential diagnosis of Kasa based on kapha	MK	161
◉ lakshana		
◉ Describe the differential diagnosis of Kshayaja kasa and Rajayakshma	MK	161
◉ Describe the differential diagnosis of Kshataja kasa and kshata ksheena	MK	160
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Topic 15 15. Rajayakshma & Shosha (Lecture :3 hours, Non lecture: 0 hours)		
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◉ Explain tri roopa, shad roopa, ekadasha roopa, and sadhyasadhyata of Rajayakshma	DK	174-178
◉ Classify and describe Ashta shosha	DK	172
opic 16 16. Common lung disorders (Lecture :1 hours, Non lecture: 4 hours)		
◉ Describe the clinical features of Pneumonia, Chronic Obstructive Pulmonary Disease, Pleural effusion, and Bronchiectasis	DK	181-192
◉ Perform relevant clinical examination of Pneumonia, Chronic Obstructive Pulmonary Disease, Pleural effusion, and Bronchiectasis	DK	181-192
◉ Order and interpret relevant investigations of Pneumonia, Chronic Obstructive Pulmonary Disease, Pleural effusion, and Bronchiectasis	DK	181-192
opic 17 17. Jwara (Lecture :4 hours, Non lecture: 1 hours)		
◉ Define and enlist types of Jwara based on vidhi samprapti	MK	200
◉ Describe nidana, samprapti, samanya poorvaroopa, vishishta poorvaroopa, pratyatma lakshana and samprapti of Doshaja jwara	MK	198-200
◉ Describe Doshaja jwara along with sannipataja jwara according to Charaka	MK	206
◉ Describe Abhishanga jwara, Vishama jwara, and Punaravartaka jwara	MK	203, 214
◉ Explain Antarvega, Bhahirvega jwara, Vata balasaka and Pralepaka jwara	MK	201,203
◉ Differentiate Ama, Pachyamana and Nirama jwara lakshana	MK	202-203
◉ Analyze Agantu and Doshaja jawara with reference to Jwara samprapti	MK	211
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Learning Objective **(At the end of the session, the students should be able to)**	**Must to know/ desirable to know / Nice to know**	**Page No.**
⦿ Describe samprapti of Masurika	NK	218
⦿ Enlist bheda of Masurika	NK	220
⦿ Explain the avasthika lakshana of Masurika	NK	223
⦿ Enlist features of Romantika	NK	225
Topic 19 19. Fever (Lecture :1 hours, Non lecture: 6 hours)		
⦿ Describe the organism, incubation period, and mode of transmission of Measles virus, Varicella-zoster virus and Herpes zoster, Coxsackie virus, Rubella virus, various Malaria parasites, Influenza virus, Dengue virus, Leptospira, Chikungunya virus, Salmonella and causative agents of other common regional disorders presenting with fever	DK	230-254
⦿ Describe the clinical features of Measles, Chicken pox and Herpes zoster, Hand foot mouth disease, Rubella, Malaria, Filariasis, Influenza, Dengue, Leptospirosis, Chikungunya, and Typhoid	DK	230-254
⦿ Describe the common regional disorders presenting with fever	DK	230-254
⦿ Describe the complications of Measles, Chicken pox and Herpes zoster, Hand foot mouth disease, Rubella, Malaria, Filariasis, Influenza, Dengue, Leptospirosis, Chikungunya, Typhoid, and other common regional disorders presenting with fever	DK	230-254
⦿ Perform relevant clinical examination related to Measles, Chicken pox and Herpes zoster, Hand foot mouth disease, Rubella, Malaria, Filariasis, Influenza, Dengue, Leptospirosis, Chikungunya, Typhoid, and other common regional disorders presenting with fever	DK	230-254
⦿ Order and interpret relevant investigations related to Measles, Chicken pox and Herpes zoster, Hand foot mouth disease, Rubella, Malaria, Filariasis, Influenza, Dengue, Leptospirosis, Chikungunya, Typhoid, and other common regional disorders presenting with fever	DK	230-254
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⦿ Enlist bheda of Pandu	MK	263
⦿ Describe purvaroopa of Pandu	MK	262
⦿ Enlist upadrava of Pandu	MK	267
⦿ Describe sadhya asadhyata of Pandu	MK	267
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⦿ Differentiate Doshaja pandu	MK	266
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⦿ Define Raktapitta and mention the rakta pravrutti marga	MK	269

Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
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⦿ Enlist bheda of Raktapitta	MK	273
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Topic 22 22. Hematopoietic diseases (Lecture :1 hours, Non lecture: 6 hours)		
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⦿ Order and interpret relevant investigations of anaemia, nutritional anaemia, thalassemia, sickle cell anaemia, leukaemia, and thrombocytopenia	DK	280-298
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⦿ Describe hetu and samprapti of Hridroga	DK	304
⦿ Enlist bheda of Hridroga	DK	305
⦿ Analyze samanya lakshana of Hridroga	DK	305
⦿ Describe vishishta lakshana of Hridroga	DK	305
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Topic 24 24. Shotha (Lecture :2 hours, Non lecture: 0 hours)		
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⦿ Enlist bheda of Shotha	MK	313
⦿ Describe vidhi samprapti of shotha	MK	313
⦿ Describe purvaroopa, vishishta lakshana, samprapti and sadhya asadhyata of Nija shotha	MK	315-319
⦿ Differentiate Doshaja shotha	MK	317
Topic 25 25. Cardiovascular disorders (Lecture :1 hours, Non lecture: 5 hours)		
⦿ Describe the clinical features of Coronary Artery Disease (Ischemic Heart Disease, and Myocardial Infarction) and Congestive cardiac failure	DK	321-327
⦿ Perform relevant clinical examination of Coronary Artery Disease (Ischemic Heart Disease, and Myocardial Infarction) and Congestive cardiac failure	DK	321-327
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Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
Topic 26 26. Kamala (Lecture :2 hours, Non lecture: 0 hours)		
⦿ Define Kamala	MK	330
⦿ Describe hetu and samprapti of Kamala	MK	330
⦿ Classify Kamala on the basis of vidhi samprapti of Kamala	MK	332
⦿ Differentiate Koshta shakhashrita and Shakhashrita Kamala	MK	333
⦿ Enlist upadrava of Kamala	MK	334
⦿ Describe sadhya asadhyata of Kamala	MK	335
⦿ Describe Swatantra-Paratantra Kamala, Alpapitta-Bahupitta Kamala	MK	332
Topic 27 27. Udara Roga (Lecture :2 hours, Non lecture: 1 hours)		
⦿ Explain pratyatma lakshana of Udara	MK	336
⦿ Describe hetu and samprapti of Udara	MK	337
⦿ Enlist bheda of Udara	MK	339
⦿ Describe purvaroopa, samanya lakshana and sadhya asadhyata of Udara	MK	349
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⦿ Differentiate Doshaja udara	MK	342
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Topic 28 28. Hepatobiliary diseases (Lecture :1 hours, Non lecture: 2 hours)		
⦿ Describe the clinical features of Liver cirrhosis, Alcoholic and Non - Alcoholic Liver Disease, Hepatitis, Jaundice and Ascites	DK	351-361
⦿ Perform relevant clinical examination of Liver cirrhosis, Alcoholic and Non - Alcoholic Liver Disease, Hepatitis, Jaundice and Ascites	DK	351-361
⦿ Order and interpret relevant investigations of Liver cirrhosis, Alcoholic and Non - Alcoholic Liver Disease, Hepatitis, Jaundice and Ascites	DK	351-361
⦿ Describe the differential diagnosis of Liver cirrhosis, Alcoholic and Non - Alcoholic Liver Disease, Hepatitis, Jaundice and Ascites	DK	351-361
Topic 29 29. Kushtha - Maha Kushtha & Kshudra Kushtha (According to Charaka) (Lecture :3 hours, Non lecture: 1 hours)		
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⦿ Describe dosha anusara Kushtha lakshana	MK	371

Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
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◉ Differentiate Maha kushtha & Kshudra kushtha	MK	379
Topic 30 30. Sheetapitta (Lecture :1 hours, Non lecture: 0 hours)		
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◉ Describe Kotha lakshana	MK	384
◉ Differentiate Sheetapitta and Udarda	MK	385
Topic 31 31. Shwitra (Lecture :1 hours, Non lecture: 0 hours)		
◉ Enlist types of Shwitra	MK	388
◉ Describe lakshana and sadhya asadhyata of Shwitra	MK	389
Topic 32 32. Visarpa (Lecture :2 hours, Non lecture: 0 hours)		
◉ Define Visarpa	MK	391
◉ Describe hetu and samprapti of Visarpa	MK	391
◉ Enlist bheda of Visarpa	MK	394
◉ Describe purvaroopa of visarpa, features of Bahya and Abhyantara visarpa and sadhya asadhyata of Visarpa	MK	394, 399
◉ Differentiate Doshaja Visarpa	MK	396
◉ Explain Agni, Kardama and Granthi visarpa	MK	396-397
Topic 33 33. Skin diseases (Lecture :1 hours, Non lecture: 6 hours)		
◉ Describe the clinical features of Allergic disorders - Eczema, Urticaria; Squamous lesions - Psoriasis, Lichen planus; Bullous lesion – Pemphigus and Pemphigoid; Mycotic skin diseases; Leprosy; Vitiligo; Cellulitis	DK	400-422
◉ Perform relevant clinical examination of Allergic disorders - Eczema, Urticaria; Squamous lesions - Psoriasis, Lichen planus; Bullous lesion – Pemphigus and Pemphigoid; Mycotic skin diseases; Leprosy; Vitiligo; Cellulitis	DK	400-422
◉ Order and interpret relevant investigations of Allergic disorders - Eczema, Urticaria; Squamous lesions - Psoriasis, Lichen planus; Bullous lesion – Pemphigus and Pemphigoid; Mycotic skin diseases; Leprosy; Vitiligo; Cellulitis	DK	400-422
Topic 34 34. Galaganda (Lecture :1 hours, Non lecture: 0 hours)		
◉ Describe pratyatma lakshana and samprapti of Galaganda	NK	428
Topic 35 35. Thyroid disorders (Lecture :1 hours, Non lecture: 1 hours)		
◉ Describe the clinical features of Hypothyroidism and hyperthyroidism	DK	436-440
◉ Perform relevant clinical examination of Hypothyroidism and hyperthyroidism	DK	436-440

Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
◉ Order and interpret relevant investigations of Hypothyroidism and hyperthyroidism	DK	436-440
Topic 36 36. Sthoulya – Karshya (Lecture :1 hours, Non lecture: 0 hours)		
◉ Describe nidana of Sthoulya and Karshya	MK	443, 447
◉ Describe samprapti of Sthoulya	MK	443
◉ Describe samanya and vishista lakshana of Sthoulya	MK	445
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Topic 37 37. Obesity (Lecture :1 hours, Non lecture: 1 hours)		
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◉ Perform relevant clinical examination of Obesity	DK	452
◉ Order and interpret relevant investigations of Obesity	DK	453
◉ Describe the differential diagnosis of Obesity	DK	453
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◉ Enlist bheda of Prameha	MK	457
◉ Describe purvaroopa, vishishta lakshana, upadrava and sadhya asadhyata of Prameha	MK	463-469
Topic 39 39. Diabetes Mellitus and Pancreatitis (Lecture :1 hours, Non lecture: 1 hours)		
◉ Describe the clinical features of Diabetes Mellitus and Pancreatitis	DK	471-475
◉ Describe the complications of Diabetes Mellitus and Pancreatitis	DK	471-475
◉ Perform relevant clinical examination of Diabetes Mellitus and Pancreatitis	DK	471-475
◉ Order and interpret relevant investigations of Diabetes Mellitus and Pancreatitis	DK	471-475
◉ Describe Pancreatitis induced Diabetes mellitus (Fibrocalculous pancreatic Diabetes)	DK	474
Topic 40 40. Vatavyadhi (Lecture :1 hours, Non lecture: 0 hours)		
◉ Describe nidana, samanya purvaroopa, and samanya lakshana of Vatavyadhi	MK	478-482
Topic 41 41. Snayugata vata (Lecture :4 hours, Non lecture: 2 hours)		
◉ Define Akshepaka	MK	484
◉ Enlist Snayugata vata	MK	483

Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
◉ Describe Akshepaka	MK	484
◉ Enlist Akshepaka bheda	MK	485
◉ Explain Akshepaka Samprapti	MK	485
◉ Describe Apatanaka	MK	485
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◉ Describe Dandapatanaka, Antarayama and Bahirayama	MK	487
◉ Define Ardita	MK	489
◉ Describe Ardita, samprapti of Ardita and lakshana of Ardita	MK	489
◉ Compare Ardita according to Charaka and Sushruta	MK	491
◉ Enlist Sadyasadyata of Ardita	MK	492
◉ Define Pakshaghata	MK	492
◉ Describe Pakshaghata samprapti, and Pakshaghata lakshana	MK	492
◉ Describe Kampavata	MK	494
◉ Describe Gridhrasi and Vishwachi with its lakshana	MK	494-495
◉ Describe differential diagnosis of Gridhrasi and Viswachi	MK	495
◉ Describe Pangutwa	MK	496
◉ Differentiate Khanja with Gridhrasi	MK	496
Topic 42 42. Common neurologic and spine disorders (Lecture :2 hours, Non lecture: 4 hours)		
◉ Describe the clinical features of Common neurologic diseases: Parkinson's disease, Stroke, Bell's Palsy, Motor Neuron Disease, Transverse myelitis, Epilepsy (Organic) and Common Spine disorders: Lumbago- Sciatica syndrome, Brachial neuralgia, Cervical and Lumber Spondylosis	DK	498-517
◉ Perform relevant clinical examination of Common neurologic diseases: Parkinson's disease, Stroke, Bell's Palsy, Motor Neuron Disease, Transverse myelitis, Epilepsy (Organic) and Common Spine disorders: Lumbago- Sciatica syndrome, Brachial neuralgia, Cervical and Lumber Spondylosis	DK	498-517
◉ Order and interpret relevant investigations of Common neurologic diseases: Parkinson's disease, Stroke, Bell's Palsy, Motor Neuron Disease, Transverse myelitis, Epilepsy (Organic) and Common Spine disorders: Lumbago- Sciatica syndrome, Brachial neuralgia, Cervical and Lumber Spondylosis	DK	498-517
Topic 43 43. Sandhigatavata and Asthi majja gata vata (Lecture :1 hours, Non lecture: 0 hours)		
◉ Describe Sandhigatavata	MK	521
◉ Describe Katigraha and Manyastambha	MK	523, 524

Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
⦿ Explain Vatakantaka	MK	524
⦿ Describe Avabahuka and Amsashosha	MK	525
⦿ Describe the differential diagnosis of Avabahuka with Vishwachi	MK	526
⦿ Describe Asthi-majjagata vata	MK	526
Topic 44 **44. Diseases of bone, joints, and muscles** **(Lecture :1 hours, Non lecture: 2 hours)**		
⦿ Describe the clinical features of Diseases of bone and Joints - Osteoarthritis, Osteoporosis, Frozen Shoulder, Calcaneal spur/ Plantar fasciitis, Tennis elbow, Carpel tunnel syndrome; Muscular diseases - Muscular Dystrophy	DK	527-538
⦿ Perform relevant clinical examination of Diseases of bone and Joints - Osteoarthritis, Osteoporosis. Frozen Shoulder, Calcaneal spur/ Plantar fasciitis, Tennis elbow, Carpel tunnel syndrome; Muscular diseases - Muscular Dystrophy	DK	527-538
⦿ Order and interpret relevant investigations of Diseases of bone and Joints - Osteoarthritis, Osteoporosis. Frozen Shoulder, Calcaneal spur/ Plantar fasciitis, Tennis elbow, Carpel tunnel syndrome; Muscular diseases - Muscular Dystrophy	DK	527-538
Topic 45 **45. Amavata** **(Lecture :2 hours, Non lecture: 0 hours)**		
⦿ Describe hetu and samprapti of Amavata	MK	543
⦿ Enlist bheda of Amavata	MK	544
⦿ Describe samanya lakshana, vishishta lakshana, upadrava and sadhya asadhyata of Amavata	MK	544-547
Topic 46 **46. Vatarakta** **(Lecture :2 hours, Non lecture: 1 hours)**		
⦿ Define Vatarakta with its synonyms	MK	548
⦿ Describe hetu and samprapti of Vatarakta	MK	549
⦿ Explain Samprapti of Avasthika Vatarakta	MK	549
⦿ Enlist and explain vidhi samprapti of Vatarakta	MK	551
⦿ Enlist and explain bheda of doshaja Vatarakta	MK	552
⦿ Describe purvaroopa, upadrava, and sadhya asadhyata of Vatarakta	MK	553-554
⦿ Describe the differential diagnosis of Sandhigata vata, Amavata and Vata rakta	MK	555
Topic 47 **47. Immunological & Metabolic disorders** **(Lecture :1 hours, Non lecture: 2 hours)**		
⦿ Describe the clinical features of Rheumatic fever, Rheumatoid arthritis, SLE, Ankylosing spondylitis, Gout	DK	556-566
⦿ Perform relevant clinical examination of Rheumatic fever, Rheumatoid arthritis, SLE, Ankylosing spondylitis, Gout	DK	556-566

Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
⊙ Order and interpret relevant investigations of Rheumatic fever, Rheumatoid arthritis, SLE, Ankylosing spondylitis, Gout	DK	556-566
⊙ Describe the differential diagnosis of Arthritis	DK	567
Topic 48 48. Klaibya & Vandhyatva (Lecture :1 hours, Non lecture: 0 hours)		
⊙ Describe Klaibya and vandhyatva	NK	571, 583
⊙ Enlist types of Klaibya and Vandhyatva	NK	574, 585
Topic 49 49. Sexual dysfunction and Infertility (Lecture :1 hours, Non lecture: 1 hours)		
⊙ Order and interpret relevant investigations of Sexual dysfunction and Infertility	NK	589-611
⊙ Describe differential diagnosis of Sexual dysfunction and Infertility	NK	589-611
Topic 50 50. Unmada & Apasmara (Lecture :3 hours, Non lecture: 0 hours)		
⊙ Explain nirukti of Unmada and Apasmara	NK	614, 626
⊙ Explain pratyatmalakshana of Unmada and Apasmara	NK	615, 626
⊙ Describe hetu and samprapti of Unmada and Apasmara	NK	616, 627
⊙ Enlist bheda of Unmada and Apasmara	NK	618, 629
⊙ Describe samanya lakshana of Unmada and Apasmara	NK	618, 629
Topic 51 51. Vishada (Lecture :1 hours, Non lecture: 0 hours)		
⊙ Define Vishada	NK	635
Topic 52 52. Murchha, and Sanyasa (Lecture :1 hours, Non lecture: 0 hours)		
⊙ Describe the Utpadaka hetu of Murchha, and Sanyasa	NK	639, 641
⊙ Describe samprapti of Murchha, and Sanyasa	NK	639
Topic 53 53. Common Psychiatric diseases (Lecture :1 hours, Non lecture: 0 hours)		
⊙ Describe clinical manifestation of Depression, Anxiety neurosis and Epilepsy (Non-organic)	NK	646-651
⊙ Differentiate Depression, Anxiety neurosis, and Epilepsy (Non- organic)	NK	646-651
⊙ Perform mental status examination for Depression, Anxiety neurosis, and Epilepsy (Non-organic)	NK	646-651
Topic 54 54. Phiranga and Upadamsha (Lecture :1 hours, Non lecture: 0 hours)		
⊙ Describe nidana of Phiranga and Upadamsha	NK	654, 656
⊙ Explain the Upadamsha samprapti	NK	656
⊙ Enlist the types of Phiranga	NK	654
Topic 55 55. Syphilis & Gonorrhoea (Lecture :1 hours, Non lecture: 1 hours)		
⊙ Describe causative factors, and clinical features of Syphilis and Gonorrhoea	NK	663-665

Learning Objective (At the end of the session, the students should be able to)	Must to know/ desirable to know / Nice to know	Page No.
⦿ Describe differential diagnosis of Syphilis and Gonorrhoea	NK	663-665
⦿ Order and interpret relevant investigation of Syphilis and Gonorrhoea	NK	663-665
Topic 56 56. Krimiroga (Lecture :1 hours, Non lecture: 0 hours)		
⦿ Describe hetu of Krimiroga	NK	669
⦿ Enlist bheda of Krimiroga	NK	670
⦿ Describe samanya lakshana of Krimiroga	NK	670
Topic 57 57. Clinical presentation of common parasitic disorders (Lecture :1 hours, Non lecture: 2 hours)		
⦿ Describe the clinical presentation of Hook worm, Round worm, Thread worm, and Pin worm	DK	678-682
⦿ Perform relevant examination of clinical presentation of Hook worm, Round worm, Thread worm, and Pin worm	DK	678-682
⦿ Order and interpret relevant investigations of clinical presentation of Hook worm, Round worm, Thread worm, and Pin worm	DK	678-682
Topic 58 58. Khalitya & Palitya (Lecture :1 hours, Non lecture: 0 hours)		
⦿ Describe Khalitya and Palitya	NK	684
Topic 59 59. Shleepada (Lecture :1 hours, Non lecture: 0 hours)		
⦿ Describe nidana and samprapti of Shleepada	NK	691
⦿ Enlist bheda of Shleepada	NK	693
⦿ Enlist features of Shleepada	NK	693
Topic 60 60. Tuberculosis (Lecture :1 hours, Non lecture: 2 hours)		
⦿ Describe the clinical presentation of Tuberculosis	DK	698
⦿ Perform relevant clinical examination of Tuberculosis	DK	700
⦿ Order and interpret relevant investigations of clinical presentation of Tuberculosis	DK	700

TOPIC 1

Agnimāndhya

Introduction

Agnimāndhya is reduction in the intensity of Agni (digestive fire). Agnimāndhya may occur as a separate disease or as a symptom in other diseases and as a main pathogenesis in many diseases. Bṛhatrayi's explained Agnimāndhya as a clinical course of other diseases, but Ācārya Caraka while explaining the Udara roga, he had described that Mandāgni is the cause for Sarvaroga (all the disorders). Ācārya Mādhavakara described Agnimāndhya as a separate disease along with Ajīrṇa because of Parasparakāraṇa (mutual causative factors) and also Mādhavakara did not highlight Mandāgni alone in Agnimāndhya, he emphasized that any type of increased or decreased functioning of Agni can be considered in the term Agnimāndhya i.e. impairment of Agni. Ācārya Cakrapāṇi supports the above statement by describing Agnidoṣoatra agnimāndyameva vivakṣitaṃ i.e. Agni doṣa (impairment of Agni) refers to Agnimāndhya (subdued digestive fire).

Importance of Agni

- आयुर्वर्णो बलं स्वास्थ्यमुत्साहोपचयौ प्रभा। ओजस्तेजोऽग्नयः प्राणाश्चोक्ता देहाग्निहेतुकाः॥
  शान्तेऽग्नौ म्रियते, युक्ते चिरं जीवत्यनामयः। रोगी स्याद्विकृते, मूलमग्निस्तस्मान्निरुच्यते॥ (Ca.Ci.15/3-4)

Ācārya Caraka described that Dehāgni (digestive fire of the body) is the root cause of Āyu (lifespan), Varṇa (complexion), Balaṃ (strength), Svāsthya (health), Utsāha (enthusiasm), Upacaya (nourishment) Prabhā (luster), Ojas, Tejas (body heat), Agni and Prāṇa (vital breathe) because Śānteagnau mriyate (if this Agni extinguishes, one become dead), Yukte ciraṃ jīvatyanāmayaḥ (if it is functioning properly the person lives long without any disease) and Rogī syādvikṛte (if this Agni become deranged, then the individual becomes diseased).

- ....दोषधातुमलसन्निपातजनितोऽन्तरुष्मा........ ॥ (A.S.Śā.6/35)

- यदन्नं देहधात्वोजोबलवर्णादिपोषकम्। तत्राग्निर्हेतुराहारान्न ह्यपक्वाद्रसादयः॥ (Ca.Ci.15/5)

Agni is the heat produced inside the body by the combination of the doṣas, dhātus and Malas. In the presence of normal functioning of Agni (digestive fire), the ingested food nourishes the Deha (body), Dhātu (tissues), Ojas, Bala (strength), Varṇa (complexion) etc. whereas, in the absence of normal functioning of Agni, the Rasādi dhātus cannot be developed from undigested food. By these one can understand that, Agni is responsible for all digestive and metabolic process in the body.

- ⊙ मरीचिरुवाच- अग्निरेव शरीरे पित्तान्तर्गतः कुपिताकुपितः शुभाशुभानि करोति; तद्यथा- पक्तिमपक्तिं दर्शनमदर्शनं मात्रामात्रत्वमूष्मणः प्रकृतिविकृतिवर्णौ शौर्यं भयं क्रोधं हर्षं मोहं प्रसादमित्येवमादीनि चापराणि द्वन्द्वानीति॥ (Ca.Sū.12/11)

Ācārya Marīci while describing the functions of Pitta, explained that the Agni which is present inside the body refers to heat of Pitta in the body which produces Śubhāśubhā (normal or abnormal) conditions in Kupitākupita (non-aggravated and aggravated) conditions respectively such as Paktimapakti (digestion or indigestion), Darśanamadarśana (vision or loss of vision), Mātrāmātratvamūṣmaṇaḥ (normalcy or otherwise heat of the body), Prakṛtivikṛtirvarṇau (normal or abnormal complexion), Śauryaṃ bhayaṃ (prowess-fear), Krodhaṃ harṣaṃ (anger- happiness), Mohaṃ prasāda (delusion-clarity) etc., other such duals. Ācārya Cakrapāṇi while commenting on Pittāntargataḥ, mentioned that the function of Pitta inside the body is not as external fire having flame, but to provide heat of Agni

- ⊙ तत्र पित्तविशेषः पाचकसंज्ञोऽग्निरिति .... । (A.S.Śā.6/34)
- ⊙ न खलु पित्तव्यतिरेकादन्योऽग्निरुपलभ्यते । (Su.Sū.21/9)
- ⊙ तच्चादृष्टहेतुकेन विशेषेण पक्वामाशयमध्यस्थं पित्तं चतुर्विधमन्नपानं पचति, विवेचयति च दोषरसमूत्रपुरीषाणि; तत्रस्थमेव चात्मशक्त्या शेषाणां पित्तस्थानानां शरीरस्य चाग्निकर्मणाऽनुग्रहं करोति, तस्मिन् पित्ते पाचकोऽग्निरिति सञ्ज्ञा; । (Su.Sū.21/10)

Even Ācārya Suśruta and Vāgbhaṭa opines the same that Pācaka Pitta which is located in between Pakvāśaya (large intestine) and Āmāśaya (stomach) is called as Agni because it does Caturvidhamannapānaṃ pacati (digests the four types of food) or Annasya paktā (digests the food) and also helps in separation of Doṣa, Rasa, Mūtra and Purīṣa from one another. Hence, it is called as Pācakāgni, Audaryāgni, Antarāgni, Kāyagni, Koṣṭhāgni, and also as Vaiśvānara.

- ⊙ न तु पित्तादभेदं, पित्तेनाग्निमान्द्यस्य ग्रहण्यध्याये वक्ष्यमाणत्वात्, तथा पित्तहरस्य सर्पिषोऽग्निवर्धनत्वेनोक्तत्वात्। (Ca.Ca.Sū.12/11)

However, Ācārya Cakrapāṇi described that Pitta and Agni are not totally identical, because Pitta kṣaya is the cause for Agnimāndhyata and Ghṛta which pacifies the Pitta is prescribed to stimulate the Agni (digestive fire).

- ⊙ अन्नस्य पक्ता सर्वेषां पक्तॄणामधिपो मतः। तन्मूलास्ते हि तद्वृद्धिक्षयवृद्धिक्षयात्मकाः॥
  तस्मात्तं विधिवद्युक्तैरन्नपानेन्धनैर्हितैः। पालयेत् प्रयतस्तस्य स्थितौ ह्यायुर्बलस्थितिः॥ (Ca.Ci.15/39-40)
- ⊙ जाठरो भगवानग्निरीश्वरोऽन्नस्य पाचकः । (Su.Sū.35/27)

- ⊙ आहारमग्निः पचति दोषानाहारवर्जितः। धातून् क्षीणेशु दोषेषु जीवितं धातुसङ्क्षये॥ (A.H.Ci.10/91)

Agni is classified in to 13 types i.e. Jaṭharāgni (one in number), Bhūtāgni (five in numbers) and Dhātvāgni (seven in numbers). Among these, Jaṭharāgni is the prime one because aggravation and diminution of Bhūtāgni and Dhātvāgni depends on Jaṭharāgni. Ācārya Suśruta described that Jaṭharāgni is itself Bhagavān (God) and is responsible for Annasya Pācakaḥ (digestion of the food). The person should take proper care to maintain the Agni by providing wholesome fuel in the form of food and drinks; because strength and longevity of the person is depend on Agni. Vāgbhaṭa described that Agni does Āhara pacati (digests the food); in absence of food, it digests the doṣas, when the doṣas become Kṣīṇā (decreased), then it digests the dhātus and when the dhātus becomes Saṅkṣaya (depleted), then it decreases the Jīvita (life span).

Nidāna and Saṃprāpti of Agnimāndhya

- ⊙ अभोजनादजीर्णातिभोजनाद्विषमाशनात्। असात्म्यगुरुशीतातिरूक्षसन्दुष्टभोजनात्॥
  विरेकवमन स्नेहविभ्रमाद्व्याधिकर्षणात्। देशकालर्तुवैषम्याद्वेगानां च विधारणात्॥
  दुष्यत्यग्निः, स दुष्टोऽन्नं न तत् पचति लघ्वपि। अपच्यमानं शुक्तत्वं यात्यन्नं विषरूपताम् ॥ (Ca.Ci.15/42-44)

Due to Abhojanāt (fasting), Ajīrṇāt (intake of food during indigestion), Atibhojanāt (overeating), Viṣamāśanāt (irregular eating habit), Asātmya bhojanāt (eating unwholesome food), Guruśītātirūkṣasaṃduṣṭabhojanāt (indulging in food which is heavy, cold, excessively non-unctuous and polluted food), Virekavamanasnehavibhramāt (improper application of purgation, emesis and oleation therapies), Vyādhikarṣaṇāt (emaciation due to other diseases), Deśakālarṭuvaiṣamyāt (due to vitiation of place, time and season) and Vegavidhārāṇāt (suppression of natural urges), the Agni gets vitiated, This vitiated Agni does not digest even the light food. Thus, the Apacyamāna anna (undigested food) produces the Śuktatva (sourness) resulting in Viṣarupatām (similar to poison) and causes various types of disorders at this stage

Saṃprāpti Ghaṭaka

Doṣa	Kapha Pradhāna Tridoṣas
Duṣya	Rasa
Agni	Jaṭharāgni Dhatvāgni māndhya
Āma	Agni janya Āma
Srotas	Rasa and Anna vaha
Srotoduṣṭi	Saṅga

Udbhava Sthāna	Āmapakvāśaya
Sañcāra Sthāna	Sarva Śārīra
Vyakta sthāna	Sarva Śārīra
Adhiṣṭhāna	Pakvāmāśaya madhye
Rogamārga	Abhyantara

Bheda and Lakṣaṇās

- मन्दस्तीक्ष्णोऽथ विषमः समश्चेति चतुर्विधः । कफपित्तानिलाधिक्यात्तत्साम्याज्जाठरोऽनलः ॥ (M.N.6/1)
- प्रागभिहितोऽग्निरन्नस्य पाचकः । स चतुर्विधो भवति- दोषानभिपन्न एकः, विक्रियामापन्नस्त्रिविधो भवति- विषमो वातेन, तीक्ष्णः पित्तेन, मन्दः श्लेष्मणा, चतुर्थः समः सर्वसाम्यादिति । (Su.Sū.35/24)

Jaṭharāgni is also classified in to four types based on the morbidities caused by abnormal Agni namely Mandāgni, Tīkṣṇāgni, Viṣamāgni and Samāgni. The first three are due to predominance of Kapha, Pitta and Vāta respectively and the fourth due to the normalcy of all the three doṣas.

- तत्र, यो यथाकालमुपयुक्तमन्नं सम्यक् पचति स समः, समैर्दोषैः; यः कदाचित् सम्यक् पचति, कदाचिदाध्मानशूलोदावर्तातिसारजठरगौरवान्त्रकूजनप्रवाहणानि कृत्वा, स विषमः; यः प्रभूतमप्युपयुक्तमन्नमाशु पचति स तीक्ष्णः, स एवाभिवर्धमानोऽत्यग्निरित्याभाष्यते, स मुहुर्मुहुः प्रभूतमप्युपयुक्तमन्नमाशुतरं पचति, पाकान्ते च गलताल्वोष्ठशोषदाहसन्तापाञ्जनयति; यस्त्वल्पमप्युपयुक्तमुदरशिरोगौरवकासश्वासप्रसेकच्छर्दिगात्रसदनानि कृत्वा महता कालेन पचति, स मन्दः ॥ (Su.Sū.35/24)
- विषमो वातजान् रोगांस्तीक्ष्णः पित्तनिमित्तजान् । करोत्यग्निस्तथा मन्दो विकारान् कफसम्भवान् ॥ (Su.Sū.35/25)

Samāgni

- समा समाग्नेरशिता मात्रा सम्यग्विपच्यते । एषां मध्ये तु सर्वेषां समाग्निः श्रेष्ठ उच्यते ॥ (Bhā.P.Ma.6/6)

The one which digests the consumed food in the proper time is called Samāgni, which maintains the equilibrium of dhātus as a result of Samaḥ samaiḥ i.e. equilibrium of doṣas helps to maintain the normalcy of Agni. This Agni is said to be Śreṣṭha (ideal).

Viṣamāgni

- अशिता खलु मात्राऽपि विषमाग्नेस्तु देहिनः । कदाचित्पच्यते सम्यक् कदाचिन्न विपच्यते ॥
  तस्याध्मानमुदावर्त शूलं जठरगैरवम् । प्रवाहणमतीसारस्तथा स्यादान्त्रकूजन् ॥ (Bhā.P.Ma.6/4-5)

The one which digests the consumed food irregularly i.e. sometimes digests the food normally and in other times, it produces Ādhmāna (distension of abdomen), Udāvarta (upward movement of Vāta), Atisāra (diarrhea), Jaṭhara gaurava (heaviness of the abdomen), Āntrakūjana (gurgling sounds in the intestines) and Pravāhaṇā (tenesmus) is called Viṣamāgni, which is due to Vāta and causes Vātaja Vikāras

Tīkṣṇāgni

- मात्रातिमात्राऽप्यशिता तीक्ष्णाग्नेः पच्यते सुखम् । अतएव हि केनापि मतस्तीक्ष्णाग्निरुत्तम् ॥ (Bhā.P.Ma.6/3)

The one which digests the large quantities of food before the scheduled time is called Tīkṣṇāgni, which is due to Pitta and causes Pittaja Vikāras. Some scholars opined that Tīkṣṇāgni is Uttama (best)

Mandāgni

- स्वल्पाऽपि नैव मन्दोग्नोर्मात्रा भुक्ता विपच्यते । छर्दिः सादः प्रसेकः स्याच्छिरोजठरगौरवम् ॥ (Bhā.P.Ma.6/2)

The one which takes long time to digest the consumed food even if in less quantity and produces Udaraśirogaurava (heaviness in the abdomen and head), Kāsa (cough), Śvāsa (dyspnoea), Praseka (salivation), Chardi (vomiting) and Gātrasadanā (lassitude in the body) is called Mandāgni, which is due to Kapha and causes Kaphaja Vikāras.

In Contemporary science,

Agni can be taken in the broadest sense, as Kāyāgni. According to their Sthāna and Karma, they are differentiated as Jaṭharāgni, Dhātvāgni, Bhūtāgni, etc. Agnimāndhya is purely Āyurvedic concept and it cannot be correlated with any specific disease in contemporary science. But can be taken as different aspects of metabolism. Here Agnivaiṣamya (either increase or decrease) is the main cause and imbalance / dysfunction of Agni at any level is the primary cause of any disease.

AJĪRṆA

Introduction

Ajīrṇa is defined as the state of incomplete process of digestion of ingested food which occurs as the result of Agnimāndhya and it leads to the formation of toxic substance 'Āma' which is the main reason for causation of all the diseases. As Ajīrṇa is due to the deranged function of Agni, Ācārya Caraka described Ajīrṇa along with Grahaṇīdoṣa because of Āśraya Āśrayī sambandha i.e. Grahaṇī (location) is Āśraya and Agni is Āśrayī (dependent). While explaining the effects of improper quantity of food i.e. Hīna (insufficient quantity of food) and Atimātra (excess quantity of food), Ācārya Vāgbhaṭa and Ācārya Suśruta described Ajīrṇa in Mātrāśitīya Adhyāya and Annapānavidhi Adhyāya in Sūtrasthāna respectively. Ācārya Mādhavakara described Ajīrṇa as a separate disease along with Agnimāndhya because of Parasparakāraṇa (mutual causative factors). And also he had described that Ajīrṇa occurs due to Āhāra vaiṣamyāt (irregularities of food) and it is the Mūla (root cause) for Rogasaṃghāta (collection of diseases) and when Ajīrṇa is cured, then the other diseases also gets cured. Apāka and Avipāka are the synonyms of Ajīrṇa. Ajīrṇa can be predominantly characterised by conditions like dyspepsia (indigestion) in modern science.

Nirūkti and Paribhāṣā

- अजीर्ण - न जीर्ण्ण नञ्समासः। अपाकः तत्पर्य्यायः । (SKD)

Ajīrṇa is a negative compound word, which means indigestion. Apāka is the synonym of Ajīrṇa.

- अजीर्णः - जठरानलमान्द्येन भुक्तान्नादरेपाके, रोगभेदः ॥ (Vācaspatyam)

That which produces indigestion of ingested food due to Jaṭhārānalamāndhya (Agnimāndhya) is called Ajīrṇa

- न जीर्यति सुखेनान्नं विकारान् कुरुतेऽपि च । तदजीर्णमिति प्राहुरतन्मूला विविधा रुजः ॥ (Si.Ni.5/245)

That which produces indigestion of comfortably ingested food and produces various disorders is known as Ajīrṇa and it is the root cause for Vividhā Rujaḥ (different kinds of diseases)

- अनात्मवन्तः पशुवद्भुञ्जते येऽप्रमाणतः । रोगानीकस्य ते मूलमजीर्णं प्राप्नुवन्ति हि ॥ (Mā.Ni.6/14)

The person who eats large quantity of food carelessly like cattle will develop this disease Ajīrṇa and it is the root cause for many other diseases.

Nidāna and Saṃprāpti

- अत्यम्बुपानाद्विषमाशनाद्वा सन्धारणात् स्वप्नविपर्ययाच्च । कालेऽपि सात्म्यं लघु चापि भुक्तमन्नं न पाकं भजते नरस्य ॥
  ईर्ष्याभयक्रोधपरिक्षतेन लुब्धेन शुग्दैन्यनिपीडितेन । प्रद्वेषयुक्तेन च सेव्यमानमन्नं न सम्यक् परिणाममेति ॥
  (Su.Sū.46/500-501)

If the person indulges in Atyambupāna (drinking large quantity of water), Viṣamāśana (consuming food irregularly), Sandhāraṇāt (suppression of the natural urges) and Svapnaviparyayā (irregular sleeping habits, i.e. loss of sleep at nights or sleeping during the day), then even the wholesome or light food taken at the proper time will not digested. And if the person is taking the food under the influence of Īrṣya (envy), Bhaya (fear), Krodha (anger), Lubdhena (be greedy), Sugdainyanipīḍitena (afflicted with Śoka (grief)), and Pradveṣayuktena ca sevyamānamannaṃ (taking food with jealous (Pradveṣa - Mātsarya)), then the food will not be digested properly.

- अभोजनादजीर्णातिभोजनाद्विषमाशनात्। असात्म्यगुरुशीतातिरूक्षसन्दुष्टभोजनात्॥
  विरेकवमनस्नेह विभ्रमाद्व्याधिकर्षणात्। देशकालर्तुवैषम्याद्वेगानां च विधारणात्॥
  दुष्यत्यग्निः, स दुष्टोऽन्नं न तत् पचति लघ्वपि। अपच्यमानं शुक्तत्वं यात्यन्नं विषरूपताम् ॥ (Ca.Ci.15/42-44)

Due to Abhojanāt (fasting), Ajīrṇāt (intake of food during indigestion), Atibhojanāt (overeating), Viṣamāśanāt (irregular eating habit), Asātmya bhojanāt (eating unwholesome food), Guruśītātirūkṣasaṃduṣṭabhojanāt (indulging in food which is heavy, cold, excessively non-unctuous and polluted), Virekavamanasnehavibhramāt (improper application of purgation, emesis and oleation therapies), Vyādhikarṣaṇāt (emaciation due to other diseases), Deśakālartuvaiṣamyāt (due to vitiation of place, time and season) and Vegavidhārāṇāt (suppression of natural urges), the Agni gets vitiated, This vitiated Agni does not digest even the light food. Thus, the Apacyamāna anna (undigested food) produces Śuktatva (sourness) resulting in Viṣarupatām (effects similar to poison) and causes various types of disorders at this stage.

- मात्रयाऽप्यभ्यवहृतं पथ्यं चान्नं न जीर्यति। चिन्ताशोकभयक्रोधदुःखशय्याप्रजागरैः॥ (Ca.Vi.2/9)

The person will not be able to digest the wholesome or easily digestible food substances even though if consumed at proper time and quantity, due to Cintā (worry), Śoka (grief), Bhaya (fear), Krodha (anger), Duḥkhaśayyā (uncomfortable bed) and Prajāgaraiḥ (vigil).

Saṃprāpti Ghaṭaka of Ajīrṇa

Doṣa	Kapha Pradhāna Tridoṣa
Dūṣya	Rasa
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhyajanya Āma
Srotas	Rasa and Anna vaha
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Mahā srotas
Udbhava sthāna	Āmāśaya
Vyakta sthāna	Sarva Śārīra
Adhiṣṭhāna	Āmāśaya
Rogamārga	Abhyantara

Sāmānya Lakṣaṇās

- विबन्धोऽतिप्रवृत्तिर्वा ग्लानिर्मारुतमूढता॥ अजीर्णलिङ्गं सामान्यं विष्टम्भो गौरवं भ्रमः। (A.H.Sū.8/30-31)

Vibandho atipravṛttirvā (obstruction or excessive elimination of faeces and urine), Glāni (exhaustion), Mārutamūḍhatā (inactivity of Vāta), Viṣṭambha (distension of abdomen), Gaurava (heaviness of abdomen) and Bhrama (giddiness) are the general features of Ajīrṇa.

- तस्य लिङ्गमजीर्णस्य विष्टम्भः सदनं तथा। शिरसो रुक् च मूर्च्छा च भ्रमः पृष्ठकटिग्रहः॥
  जृम्भाऽङ्गमर्दस्तृष्णा च ज्वरश्छर्दिः प्रवाहणम्। अरोचकोऽविपाकश्च ......॥ (Ca.Ci.15/45-46)

Viṣṭambha (absence of movement of food), Sadana (malaise), Śiraso ruk (headache), Mūrcchā (fainting), Bhrama (giddiness), Pṛṣṭhakaṭi graham (stiffness in back and waist), Jṛmbhā (yawning), Aṅgamarda (body ache), Tṛṣṇā (thirst), Jvara (fever), Chardi (vomiting), Pravāhaṇa (tenesmus), Arocaka (anorexia) and Avipāka (indigestion) are the general features of Ajīrṇa.

- संसृज्यमानं पित्तेन दाहं तृष्णां मुखामयान्। जनयत्यम्लपित्तं च पित्तजांश्चापरान् गदान्॥
  यक्ष्मपीनसमेहादीन् कफजान् कफसङ्गतम्। करोति वातसंसृष्टं वातजांश्च गदान् बहून्॥
  मूत्ररोगांश्च मूत्रस्थं कुक्षिरोगान् शकृद्गतम्। रसादिभिश्च संसृष्टं कुर्याद्रोगान् रसादिजान्॥ (Ca.Ci.15/47-49)

If Ajīrṇa is associated with Pitta, then it produces Dāha (burning sensation), Tṛṣṇā (thirst), Mukhāmayān (diseases of mouth), Amlapitta and other Pittaja disorders; if it is associated with Kapha, then it produces Yakṣmā (tuberculosis), Pīnasa (coryza), Mehā (diabetes) and other Kaphaja disorders and if it is associated with Vāta, then it produces many Vātaja disorders. If it is localized in the Mūtrasthaṃ (urinary system), it produces Mūtra rogās (urinary disorders); similarly it produces Kukṣi rogā (abdominal disorders) if it is localized in the Śakṛt (faeces) and causes disorders of Rasādi dhātu, if it gets mixed with Rasādi dhātus.

Bheda and Lakṣaṇās

- आमं विदग्धं विष्टब्धं कफपित्तानिलैस्त्रिभिः । अजीर्णं केचिदिच्छन्ति चतुर्थं रसशेषतः ॥ (Su.Sū.46/499)
- आमं विदग्धं विष्टब्धं कफपित्तानिलैस्त्रिभिः । अजीर्णं केचिदिच्छन्ति चतुर्थं रसशेषतः ॥
  अजीर्णं पञ्चमं केचिन्निर्दोषं दिनपाकि च । वदन्ति षष्ठं चाजीर्णं प्राकृतं प्रतिवासरम् ॥ (M.N.6/5-6)

Ajīrṇa is classified into three types namely Āmājīrṇa, Vidagdhājīrṇa and Viṣṭabdhājīrṇa caused by Kapha, Pitta and Vāta doṣa respectively, some scholars enumerate the fourth type as Rasaśeṣājīrṇa (indigestion at the level of Āhāra rasa). Whereas Ācārya Mādhavakara classified Ajīrṇa into six types namely Āmājīrṇa, Vidagdhājīrṇa, Viṣṭabdhājīrṇa, Rasaśeṣājīrṇa, Dinapāki ajīrṇa and Prativāsara ajīrṇa.

Āmājīrṇa

- माधुर्यमन्नं गतमामसञ्ज्ञं, ...। (Su.Sū.46/502)
- तत्रामे गुरुतोत्क्लेदः शोथो गण्डाक्षिकूटगः । उद्गारश्च यथाभुक्तमविदग्धः प्रवर्तते ॥ (M.N.6/10)
- अजीर्णं च कफादामं तत्र शोफोऽक्षिगण्डयोः। सद्योभुक्त इवोद्गारः प्रसेकोत्क्लेशगौरवम्॥ (A.H.Sū.8/25)

In Āmājīrṇa, the food attains Mādhuryata (sweetness) and produces Gurutva (heaviness of the abdomen), Utkleda (nausea), Śotho gaṇḍākṣikūṭa (swelling of the cheeks and eyes), and

Udgāraśca yathābhuktamavidagdhaḥ pravartate (belching similar to those occurring soon after meals) and it occurs as a result of Kapha Prakopa (aggravation of Kapha doṣa).

Vidagdhājīrṇa

- ....विदग्धसञ्ज्ञं गतमम्लभावम् । किञ्चिद्विपक्वं,.........॥ (Su.Sū.46/502)
- विदग्धे भ्रमतृण्मूर्छाः पित्ताच्च विविधा रुजः । उद्गारश्च सधूमाम्लः स्वेदो दाहश्च जायते ॥ (M.N.6/11)
- पित्ताद्विदग्धं तृण्मोहभ्रमाम्लोद्गारदाहवत्॥ (A.H.Sū.8/26)

In Vidagdhājīrṇa, the food becomes Amlabhāva (sour) and Vipakva (slightly digested) and it is characterized by Bhrama (giddiness), Tṛṇ (thirst), Mūrcchā (fainting), Udgāraṣca sadhūmāmla (hot and sour belchings), Sveda (excessive perspiration), Dāha (feeling of burning sensation inside the body) and Pittāñca vividhā ruja (Oṣacoṣādi symptoms of Pitta).

Viṣṭabdhājīrṇa

- भृशतोदशूलं विष्टब्धमानद्धविरुद्धवातम् ॥ (Su.Sū.46/502)
- विष्टब्धे शूलमाध्मानं विविधा वातवेदनाः । मलवाताप्रवृत्तिश्च स्तम्भो मोहाऽङ्गपीडनम् ॥ (M.N.6/12)
- विष्टब्धमनिलाच्छूलविबन्धाध्मानसादकृत्। (A.H.Sū.8/26)

In Viṣṭabdhājīrṇa, there will be Bhṛśatodaśula (severe pricking pain and colic), Viṣṭabdhamānaddha viruddhavātam (Vāta moves opposite direction due to obstruction), Ādhmāna (distension of abdomen), Malavātāpravṛtti (non movement of faeces and flatus), Stambha (stiffness), Moha (delusion), Aṅgapīḍana (body ache) and Vividha Vātavedanā (Toda bhedādi symptoms of Vāta)

Rasaśeṣājīrṇa

- उद्गारशुद्धावपि भक्तकाङ्क्षा न जायते हृद्गुरुता च यस्य । रसावशेषेण तु सप्रसेकं चतुर्थमेतत् प्रवदन्त्यजीर्णम् ॥ (Su.Sū.46/503)
- रसशेषेऽन्नविद्वेषो हृदयाशुद्धिगौरवे । (M.N.6/12)
- अश्रद्धा हृद्व्यथा शुद्धेऽप्युद्गारे रसशेषतः। (A.H.Sū.8/29)

In Rasaśeṣājīrṇa, there will be Bhaktakāṅkṣā na jāyate or Annadveṣa (absence of desire for food or aversion to food), Aśraddhā (Annānabhilāṣa – aversion to food), Hṛdgurutā (heaviness in the cardiac region), Hṛdayāśuddhi (impurity in the heart) and Praseka (salivation) even though the person gets Udgāraśuddhi (pure belching).

Dinapāki Ajīrṇa

- निर्दोषम्आध्मानादिदुष्टेरकारकम्।दिनपाकिचेत्यहोरात्रेणाहारःपच्यतेइत्युत्सर्गः,यत्रतुमात्राकालासात्म्यादिदोषादपरदि
  पच्यते तद्दिनपाकि। (Ma.M.N.6/5-6)

In which, the digestion of food completes within Ahorarātra (day and night) or Aparadine (another or next day) due to Mātrakālāsātmyādi doṣas (defect in the quantity, timings and wholesomeness of the food) is called Dinapāki Ajīrṇa. And it is Nirdoṣa (harmless); hence it does not produce Ādmānādiduṣṭi (distension of abdomen etc., symptoms)

Prativāsara Ajīrṇa

- ⊙ प्राकृतं प्रतिवासरमिति प्राकृतमवैकारिकं, प्रतिवासरं प्रतिदिनं क्रियमाणम्। क्षुत्पिपासामलोत्सर्गादिर्जीर्णलक्षण स्यानुदयात्; तस्मादजीर्णं, तच्चाध्मानादिकं न करोतीति पूर्वेभ्यो भिन्नम्। तस्य चाभिधानप्रयोजनम् पाकार्थं वामपार्श्वशयनाद्याचारसेवा॥ (Ma.M.N.6/5-6)

In which, the digestion of food occurs normally as routine as daily with Jīrṇa lakṣaṇās like Kṣut (hunger), Pipāsa (thirst) and Malautsarga (expulsion of faeces) etc. and without producing Ajīrṇa lakṣaṇās like Ādhmāna etc., is known as Prativāsara Ajīrṇa. Still, it is one among the type of Ajīrṇa because it causes stasis of food in the Vāmapārśva due to Śayanādyācāra sevā (resorting activities like sleeping etc.)

Ajīrṇa Upadrava

- ⊙ मूर्च्छा प्रलापो वमथुः प्रसेकः सदनं भ्रमः । उपद्रवा भवन्त्येते मरणं चाप्यजीर्णतः ॥ (Su.Sū.46/504)

Mūrcchā (fainting), Pralāpā (delirium), Vamathu (vomiting), Praseka (excessive salivation), Sadana (malaise) and Bhrama (giddiness) are the complications of Ajīrṇa and even it leads to Maraṇa (death).

In Contemporary science,

Ajīrṇa can be predominantly characterised by conditions like dyspepsia (indigestion) in contemporary science. (Please refer Topic 8)

ĀNĀHA, ĀṬOPA AND ĀDHMĀNA

Introduction

Ānāha, Āṭopa and Ādhmāna each occur as a separate disease or as a symptom in other diseases. Even though Ānāha, Āṭopa and Ādhmāna are described as Annavaha sroto vikāras, but Ācārya Bhāvamiśra described these diseases under the Vātaja Nānātmaja vyādhis. Ācārya Suśruta described Ānāha along with Visūcika because of Visūcikāstulyacikitsitatvāt (similarity in the treatment) and Visūcikopdravāccānuktam (unsaid complication of Visūcikā). Ācārya Caraka described Ānāha in the Triśothiya Adhyāya and also in Trimarmīya Cikitsā Adhyāya along with Udāvarta. Ācārya Suśruta described Ādhmāna under Vātavyadhi Cikitsā and the disease Āṭopa is mentioned along with Ādhmāna. Ācārya Ḍalhaṇa opines that Udarapūra (fullness of the abdomen) is the common feature in all the three diseases, however their distinguishing features are: in Ānāha, there will be fullness of abdomen with retention of urine and faeces; in Ādhmāna, there will be fullness of abdomen with Vāta; and in Āṭopa, there will be fullness of abdomen with gurgling sound.

ĀNĀHA

Nirūkti and Paribhāṣā

- ⊙ आनाहः, मूत्रपुरीषरोधकरोगः । तत्पर्य्यायः । विबन्धः विष्टम्भः मलरोधनः । (SKD)
- ⊙ आनाहो वातमूत्रपुरीषादिनिरोधः। (Ḍa.Su.Sū.26/10)

The disease which produces Mūtrapurīṣarodha (obstruction of urine and faeces) or Vāta Mūtrapurīṣādi nirodhaḥ (obstruction of flatus, urine, faeces etc.) is known as Ānāha. Vibandha, Viṣṭambha and Malarodhana are the synonyms of Ānāha.

- ⊙ आनाह उदरपूरणलक्षणः स च वातोत्कटदोषैर्जनितः ॥ (Āḍ.Śā.Pra.7/49)

The disease which produces the features of Udarapūraṇa (fullness of the abdomen) due to aggravation of Vāta is called Ānāha.

- ⊙ आनाहाध्मानभेदः ॥ (Kā.Śā.Pra.7/49)

Ācārya Kāśirāma described that the disease Ānāha is a type of Ādhmāna (distension of abdomen).

- ⊙ आनाहो बद्धोदरता:॥ (He.A.H.Sū.6/146)
- ⊙ आनाहो जठराध्मानम् ॥ (Aru.A.H.Sū.28/8)

That which causes Baddhodaratā (Ānaddhodara - obstruction in the abdomen) or Jaṭharādhmāna (distention of abdomen) is known as Ānāha.

- ⊙ आनाह्यते आध्मानयुक्तो भवति ॥ (Ḍa.Su.U.56/7-8)

Ācārya Ḍalhaṇa opines that the disease Ānāha is nothing but that which is associated with Ādhmāna (distension of abdomen).

- ⊙ यस्य वातः प्रकुपितः कुक्षिमाश्रित्य तिष्ठति । नाधो व्रजति नाप्यूर्ध्वमानाहस्तस्य जायते॥ (Ca.Sū.18/32)

The aggravated Vāta that gets localized in the Kukṣi (abdomen) does not move either upwards or downwards and leads to the disease Ānāha.

Nidāna and Saṃprāpti

- ⊙ आमं शकृद्वा निचितं क्रमेण भूयो विबद्धं विगुणानिलेन ॥ प्रवर्तमानं न यथास्वमेनं विकारमानाहमुदाहरन्ति । (Su.U.56/20-21)

The Āmā (undigested food) or Śakṛt (faeces) gradually accumulate in the Āmāśaya (stomach) and Pakvāśaya (intestines) respectively and later gets obstructed by the Viguṇa vāta (inefficient or deficient Vāta) resulting in non-expulsion in their normal passage. This condition is known as Ānāha.

Saṃprāpti Ghaṭaka

Doṣa	Vāta
Dūṣya	Rasa
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa, Anna and Purīṣa vaha
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Mahā srotas
Udbhava sthāna	Āmapakvāśaya
Vyakta sthāna	Sarva Śarīra
Adhiṣṭhāna	Āmapakvāśaya
Rogamārga	Abhyantara

Bheda and Lakṣaṇās

- आनाहो द्विविधो ज्ञेय एकः पक्वाशयोद्भवः । आमाशयोद्भवश्चान्यः प्रत्यानाहः से कथ्यते ॥ (Śā.Pra.7/49)

Ānāha is classified in to two types based on the Udbhava sthāna (place of origin) namely Pakvāśayodbhava Ānāha (Purīṣaja Ānāha) and Āmāśayodbhava Ānāha (Āmaja Ānāha or Pratyānāha).

- साटोपमत्युग्ररुजमाध्मानमुदरे भृशम्। ऊर्ध्वाधोवातरोधेन तमानाहं प्रचक्षते॥ (A.H.Ni.11/60)

In Ānāha, there will be Atyugra udara ruja (severe abdominal pain) associated with Ādhmāna (distention of abdomen) and Āṭopa (gurgling sound in the abdomen) due to Ūrdhvādho Vātarodhana (obstruction of the upward and downward movement of Vāta).

Āmaja Ānāha or Pratyānāha

- तस्मिन् भवन्त्यामसमुद्भवे तु तृष्णाप्रतिश्यायशिरोविदाहाः ॥ आमाशये शूलमथो गुरुत्वं हृल्लास उद्गारविघातनं च । (Su.U.56/21-22)

The clinical features of Āmasamudbhava Ānāha (Āmaja ānāha or Pratyānāha) are Tṛṣṇā (thirst), Pratiśyāya (coryza), Śirovidāha (burning sensation in the head), Āmāśaya śūla (pain in the stomach), Gurutva (heaviness in the abdomen), Hṛllāsa (nausea) and Udgāravighātana (obstruction to eructation).

Purīṣaja Ānāha

- स्तम्भः कटीपृष्ठपुरीषमूत्रे शूलोऽथ मूर्च्छा सशकृद्वमेच्च ॥ श्वासश्च पक्वाशयजे भवन्ति लिङ्गानि चात्रालसकोद्भवानि । (Su.U.56/22-23)

The clinical features of Purīṣaja Ānāha are Kaṭipṛṣṭha stambha (stiffness in the back and waist), Puriṣamūtre stambha (retention of faeces and urine), Śūla (colic), Mūrcchā (fainting), Śakṛtvame (faecal vomiting), Śvāsa (dyspnoea) and Alasaka liṅgāni (the features of Alasaka).

ĀDHMĀNA AND ĀṬOPA

Āṭopa is not described separately in Āyurveda Saṃhitās and the Nidāna, Saṃprāpti and Cikitsā of Āṭopa is similar to Ādhmāna. Hence, Ādhmāna and Āṭopa are described together in this chapter.

Nirūkti and Paribhāṣā of Ādhmāna

- उदरे वायोरतीव संचय अनिलपूरणम् ॥ (Ā.Śa.Ko)

That which causes Anilapūraṇa (fullness of abdomen with Vāta) due to accumulation of Vāta is known as Ādhmāna.

- वायुना कुपितेन नाभेरधः सशूलमापूर आध्मानः। (In.A.S.Sū.5/8)

That which causes Nabheradhaḥ saśulamāpūraḥ (pain and filling up of Vāta below the umbilicus) due to aggravation of Vāta is known as Ādhmāna.

- अनिलश्च रुद्धमार्गतया आध्मानं करोतीति। (Ca.Ca.Si.7/22)

That disease Ādhmāna occurs due to Anilaśca Ruddhamārgata (obstruction in the passage of Vāta).

- उदरस्याध्मानम् वायुना परिपूरणम् ॥ (Ā.Śa.Ko)
- आध्मानं-वातेनोदरपूरणम्। (He.A.H.Sū.11/13)

That which causes Vāyunā paripūraṇa or Vātenodarapūraṇa (fullness of abdomen with Vāta) is known as Ādhmāna.

Nirūkti and Paribhāṣā of Āṭopa

- ⊙ आटोपः गुडगुडाशब्दः। (Ca.Ca.Ci.14/21)
- ⊙ आटोपः गुडगुडाशब्दविशेषो वातजन्यः ॥ (Ca.Ca.Ni.8/6-7)

That which produces Guḍguḍā śabda (gurgling sound in the abdomen) as a result of Vāta prakopa is known as Āṭopa.

- ⊙ आटोपः कुक्षौ शब्दवद्वातभ्रमणम्। (Ca.Ca.I.5/12-13)

That which produces Kukṣau śabdadvātabhramaṇam (movement of Vāta with gurgling sound in the abdomen) is known as Āṭopa.

- ⊙ आटोप आध्मानम् ॥ (Ḍa.Su.Ci.8/8) / (Ḍa.Su.Ni.2/8)
- ⊙ आटोप उदरस्याध्मानं । (Ḍa.Su.Sū.46/474)

The disease Āṭopa refers to Udarasyādhmāna (distension of abdomen)

- ⊙ आटोप उदरापुराः। (Ḍa.Su.U.42/8)

The disease Āṭopa refers to Udarāpurā (fullness of abdomen)

- ⊙ आटोपं सञ्चलनं, तेन सह वर्तत इति साटोपम्। (Ḍa.Su.Ni.1/88)
- ⊙ आटोपश्चलचलनमिति ॥ (Ma.M.Ni.22/28)

The disease Āṭopa means Sañcalana or Calacalana (movement of Vāta) along with gurgling sound in the abdomen.

- ⊙ आटोपः क्षोभः ॥ (Aru.A.H.Sū.28/8)
- ⊙ आटोपः आध्मानं क्षोभमित्यन्ते ॥ (Ḍa.Su.Sū.15/15)

The disease Āṭopa refers to Ādhmāna (distension of abdomen), some refer it as Kṣobha (irritation or trembling).

- ⊙ आटोपः रुजापूर्वकक्षोभः ॥ (Ḍa.Su.Ni.9/20)

That which produces Rujāpūrvaka kṣobha (irritating pain in the abdomen) is known as Āṭopa.

- ⊙ आटोपः सतोदो वातसंक्षोभः ॥ (Ḍa.Su.Sū.26/10)

That which produces Satodo vātasaṃkṣobha (movement of Vāta with pricking pain in the abdomen) is known as Āṭopa.

Nidāna and Saṃprāpti

- ⊙ आध्मानमिति जानीयाद्घोरं वातनिरोधजम् ॥ (Su.Ni.1/88)

Ācārya Suśruta explains that Ādhmāna occurs due to Vātanirodhajam (obstruction of flatus). Ācārya Caraka had mentioned Ādhmāna as one of Vamanavirecana vyāpat. Hence the Nidāna and Saṃprāpti of Ādhmāna which occurs as a complication of Vamana and Virecana are quoted here:

- ⊙ बहुदोषस्य रूक्षस्य हीनाग्नेरल्पमौषधम्। सोदावर्तस्य चोत्क्लिश्य दोषान्मार्गान्निरुध्य च॥ (Ca.Si.6/58)

If Alpamauṣadham (small dose of medicine) is given in the person who has Bahu doṣa (excess morbid doṣas), Rūkṣasya (non-unctuous body), Hīnāgni (less digestive fire) and Udāvartasya (suffering from upward movement of Vāta), then it leads to further aggravation of doṣas and it may cause obstruction to the passages and produces the disease Ādhmāna.

Saṃprāpti Ghaṭaka of Ādhmāna and Āṭopa

Saṃprāpti Ghaṭaka	Ādhmāna	Āṭopa
Doṣa	Vāta	Vāta
Dūṣya	Rasa	Rasa
Agni	Jaṭharāgni māndhya	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa and Anna vaha	Rasa and Anna vaha
Srotoduṣṭi	Saṅga	Saṅga, Vimārga gamana
Sañcāra sthāna	Mahā srotas	Mahā srotas
Adhiṣṭhāna	Udara	Udara
Udbhava sthāna	Āmapakvāśaya	Āmapakvāśaya
Vyakta sthāna	Sarva śārīra	Sarva śārīra
Rogamārga	Abhyantara	Abhyantara

Lakṣaṇās

- ⊙ साटोपमत्युग्ररुजमाध्मातमुदरं भृशम् । आध्मानमिति जानीयाद्घोरं वातनिरोधजम् ॥ (Su.Ni.1/88)

In Ādhmāna, the obstructed Vāta causing Atyugra ruja (severe pain) in the Ādmātamudaraṃ (distension of abdomen) associated with Āṭopa (gurgling sound).

- ⊙ भृशमाध्मापयेन्नाभिं पृष्ठपार्श्वशिरोरुजम्। श्वासविण्मूत्रवातानां सङ्गं कुर्याच्च दारुणम्॥ (Ca.Si.6/59)

Bhṛśamādhmāpayennābhiṃ (severe abdominal distension in the umbilical region), Pṛṣṭhapārśvaśirorujam (pain in the back, flanks and head), Śvāsa (dyspnoea) and Viṇmūtravātānāṃ saṅgaṃ (retention of faeces, urine and flatus) are the features of Ādhmāna occurring due to complication of Vamana and Virecana.

In Contemporary science,

Ānāha, Āṭopa and Ādhmāna can be predominantly characterised by conditions like tympanites (is also known as meteorism) in contemporary science. Tympanites is a condition characterised by accumulation of gas in the gastrointestinal tract which causes a sensation of bloating and abdominal distension. Meteorism is a common symptom of many diseases, including ordinary constipation, neurosis, chronic colitis, peritonitis and intestinal obstruction. It can be manifested as a result of swallowing too much air while eating or excessive fermentation caused by bacteria of the intestinal flora, bowel obstruction, renal stones, functional disorder, bacterial overgrowth, inflammation of the bowel, blunt kidney trauma, peritonitis and idiopathic.

Clinical features: The features of meteorism are bloated sensation, abdominal discomfort/ pain, abdominal heaviness and bursting, hiccups excessive belching, flatulence, bilious vomiting and loss of appetite along with abdominal distension.

Diagnosis: Diagnosis is based on the clinical evaluation. Abdominal examination reveals marked tenderness and tympanic node on palpation. Abdominal auscultation may reveal rumbling bowel sounds. Imaging studies like X-ray helps to identify the dilated bowel loops, air-fluid levels or free air.

MCQ's

1. What is Agnimāndhya caused by Vāta known as
a) Tīkṣṇāgni
b) Mandāgni
c) Viṣamāgni
d) All of the above

2. According to Bhāvaprakāśa, which Agni is Uttama
a) Tīkṣṇāgni
b) Mandāgni
c) Viṣamāgni
d) Samāgni

3. According to Caraka, the Āśraya Āśrayī sambandha of Ajīrṇa is
a) Agnimāndhya
b) Grahaṇīdoṣa
c) Atisāra
d) Arśas

4. Viṣṭabdhājīrṇa is caused by which doṣa
a) Vāta
b) Pitta
c) Kapha
d) Sannipātaja

5. सधूमाम्ल उद्गार is seen in
a) Āmājīrṇa
b) Vidagdhājīrṇa
c) Viṣṭabdhājīrṇa
d) Rasaśeṣājīrṇa

6. हृदयाशुद्धिगौरवे is seen in
a) Āmājīrṇa
b) Vidagdhājīrṇa
c) Viṣṭabdhājīrṇa
d) Rasaśeṣājīrṇa

7. सद्योभुक्त इवोद्गारः is seen in
a) Āmājīrṇa
b) Vidagdhājīrṇa
c) Viṣṭabdhājīrṇa
d) Rasaśeṣājīrṇa

8. In which type of Ajīrṇa, Jīrṇa lakṣaṇās like Kṣut, Pipāsa and Malautsarga is seen
a) Dinapāki Ajīrṇa
b) Rasaśeṣājīrṇa
c) Prativāsara Ajīrṇa
d) Both a and b

9. The feature of Ajīrṇa Upadrava include all except
a) Mūrcchā
b) Pralāpa
c) Vamathu
d) Pipāsa

10. Baddhodaratā is caused by
a) Ajīrṇa
b) Ānāha
c) Āṭopa
d) Ādhmāna

11. Guḍguḍā śabda is seen in
a) Ajīrṇa
b) Ānāha
c) Āṭopa
d) Ādhmāna

12. Udarapūra is the feature of
a) Ānāha
b) Āṭopa
c) Ādhmāna
d) All of the above

13. Āmaja and Purīṣaja are types of
a) Ānāha
b) Āṭopa
c) Ādhmāna
d) All of the above

14. What is tympanites (meteorism)?
a) Accumulation of gas in the respiratory tract
b) Accumulation of gas in the gastrointestinal tract
c) Accumulation of fluid in the lungs
d) Accumulation of blood in the abdomen

15. *Which condition is not commonly associated with meteorism*
    - *a) Ordinary constipation*
    - *b) Asthma*
    - *c) Chronic colitis*
    - *d) Intestinal obstruction*
16. *What is a common clinical feature of meteorism*
    - *a) Persistent cough*
    - *b) Joint pain and swelling*
    - *c) Bloated sensation and abdominal distension*
    - *d) Rash and itching*

Answer Keys

1	c	2	a	3	b	4	a	5	b	6	d	7	a	8	c
9	d	10	b	11	c	12	d	13	a	14	b	15	b	16	c

Short Answer Questions

1. *Write the importance of Agni*
2. *Explain Agnimāndhya*
3. *Explain Nidāna and Samprāpti of Ajīrṇa*

Long Answer Questions

1. *Describe the Nidāna, Samprāpti, Bheda, and Lakṣaṇās of Ajīrṇa*
2. *Define and differentiate Ānāha, Āṭopa and Ādhmāna and explain the Nidāna, Samprāpti, Bheda, and Lakṣaṇās of Ādhmāna*

◆

TOPIC 2

Chardi

Introduction

Chardi is nothing but vomiting and it may occur as a separate disease or as a symptom in other diseases. Ācārya Caraka described Chardi after Atisāra because there is two major orifices (anus and mouth) in the body to eliminate the morbid doṣas, so after describing the expulsion of morbid doṣas through anus i.e. Atisāra, he described the elimination of morbid doṣas through mouth i.e. Chardi. Ācārya Suśruta described Chardi after Tṛṣṇā because of Tṛṣṇopacāra tulyatvā (similarity in the treatment) and Tṛṣṇāpratiṣedhe charderupādānāt (Chardi is one among the line of treatment in Tṛṣṇā). Ācārya Vāgbhaṭa explained Chardi along with Rājayakṣmā because it is one of the symptoms of Rājayakṣmā. Pracchardikā, Chardam, Vamathu, Vami, Chardikā, Vānti, Udgāra, Utkārikā and Chardanam are the synonyms of Chardi.

Nirūkti and Paribhāṣā

- छर्दिः छर्दयत्यनेनेति । वमिरोगा ॥ (SKD)

That which causes Chardayati (to spit or vomit) is known as Chardi or Vamirogāḥ

- छर्दिवमनविशेषः । वेगैः कृत्वा आननं छादयतीति छर्दिः ॥ (Āḍ.Śā.P.7/28)

That which causes expelling the Chādayatī (concealed morbid doṣas) through Ānana (the mouth) with great force is known as Chardi, which is a unique form of Vamana.

- ऊर्ध्वातिदोषप्रवृत्तिरूपा छर्दिरुच्यते । (Ca.Ca.Ci.20/1)

That which causes the elimination of aggravated doṣas through upward orifice i.e. mouth is called Chardi.

- छादयन्नाननं वेगैरर्दयन्नङ्गभञ्जनैः। निरुच्यते छर्दिरिति दोषो वक्त्राद्विनिश्चरन् ॥ (Su.U.49/6)
- छादयति मुखमर्दयति च अङ्गानि स छर्दिरिति निरुच्यते । (Āt.M.N.15/1-4)

That which causes Chādayannānanaṃ (Pūrayan – fills up the mouth) with the morbid doṣas and expels the doṣas through the mouth with great force along with Aṅga bhañjana (tearing pain in the body) is known as Chardi.

Sāmānya Nidāna and Saṃprāpti

- अतिद्रवैरतिस्निग्धैरहृद्यैर्लवणैरति। अकाले चातिमात्रैश्च तथाऽसात्म्यैश्च भोजनैः॥
  श्रमात् क्षयात्तथोद्वेगादजीर्णात् कृमिदोषतः । नार्याश्चापन्नसत्त्वायास्तथाऽतिद्रुतमश्नतः ।
  अत्यन्तामपरीतस्य छर्देर्वै सम्भवो ध्रुवम् । (बीभत्सैर्हेतुभिश्चान्यैर्द्रुतमुत्क्लेशितो बलात्) (Su.U.49/3-5)

Indulging in Atidrava (excessive use of liquid), Atisnigdha (excessive use of unctuous substances), Ahṛdya (unpleasant substances), Atilavaṇa (excessive use of salts), Akāle bhojana (untimely intake of food), Atimātre bhojane (excessive intake of food), Asātmya bhojane (intake of unwholesome food), Śramāt (physical exertion), Kṣayāt (emaciation), Udvegāt (anxiety), Ajīrṇāt (indigestion), Kṛmidoṣa (worm infestations), Nāryāścāpannasattvāyā (pregnancy in women), Atidrutamaśnataḥ (intake of food quickly), Atyantāmaparītasya (excessive accumulation of Āma) and Bībhatsai (disgusting smells and sights) definitely produces the disease Chardi.

- दोषानुदीरयन् वृद्धानुदानो व्यानसङ्गतः । ऊर्ध्वमागच्छति भृशं विरुद्धाहारसेवनात् । (Su.U.49/7)

Due to Viruddhāhāra sevanāt (intake of incompatible food), the doṣas aggravates and brings the Udāna Vāta along with Vyāna vāta to the Ūrdhvā bhāga (upper part of the body) and expels through the mouth forcefully.

Saṃprāpti Ghaṭaka of Chardi

Doṣa	Kapha Vāta
Dūṣya	Rasa
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa and Anna vaha
Srotoduṣṭi	Saṅga, Vimārga gamana and Atipravṛtti
Sañcāra sthāna	Ūrdhva Śārīra Rasāyanī's
Udbhava sthāna	Āmāśaya
Vyakta sthāna	Vaktrà or Ānana
Adhiṣṭhāna	Āmāśaya
Rogamārga	Abhyantara

Pūrvarūpa of Chardi

- प्रसेको हृदयोत्क्लेशो भक्तस्यानभिनन्दनम् । पूर्वरूपं मतं छर्द्या यथास्वं च विभावयेत् ॥ (Su.U.49/8)
- तासां हृदुत्क्लेशकफप्रसेकौ द्वेषोऽशने चैव हि पूर्वरूपम् ॥ (Ca.Ci.20/6)
- तासूत्क्लेशास्यलावण्यप्रसेकारुचयोऽग्रगाः॥ (A.H.Ni.5/31)

Praseka or Kapha praseka (salivation), Hṛdayotkleśa (nausea), Āsyalāvaṇya (salt taste in the mouth) and Bhaktasyānabhinandanam or Dveṣāśana (dislike or aversion for food) are the premonitory symptoms of Chardi, which manifest according to the respective doṣas

Chardi Bheda

- दोषैः पृथक्त्रिप्रभवाश्चतस्रो द्विष्टार्थयोगादपि पञ्चमी स्यात् ॥ (Ca.Ci.20/6)

Chardi is classified into five types namely, first three types caused by doṣas each i.e. Vātaja, Pittaja and Kaphaja, the fourth one is Sannipātaja (combination of all the three doṣas) and the fifth one is Dviṣṭārthayogaja (contact with disliked objects). Ācārya Suśruta classified Chardi into five types namely Vātaja, Pittaja, Kaphaja, Sannipātaja and Āgantuja. Āgantuja Chardi further classified into five types namely Bibhatsajā, Dauhṛdajā, Āmaja, Sātmyaprakopaja and Kṛmijā

- छर्दयः सप्तधा मताः । त्रिभिर्दोषैः पृथक्तिस्राः कृमिभिः सन्निपाततः ॥ घृणया च तथा स्त्रीणां गर्भाधानाज्च जायते ॥ (Śā.P.7/28)

Whereas Ācārya Śārṅgadhara classified Chardi into seven types namely, first three types caused by three doṣas each i.e. Vātaja, Pittaja and Kaphaja, the fourth one is Kṛmija, the fifth one is Sannipātaja (combination of all the three doṣas), the sixth one is Ghṛṇaja and the seventh one is Garbhādhānāja.

Viśeṣa Nidāna, Saṃprāpti and Lakṣaṇās

Vātaja Chardi

- व्यायामतीक्ष्णौषधशोकरोगभयोपवासाद्यतिकर्शितस्य । वायुर्महास्रोतसि सम्प्रवृद्ध उत्क्लेश्य दोषांस्तत ऊर्ध्वमस्यन् ।
  आमाशयोत्क्लेशकृतां च मर्म प्रपीडयंश्छर्दिमुदीरयेत्तु । (Ca.Ci.20/7-8)

In the person who had become Atikarśita (excessively emaciated) due to Vyāyāma (excessive exercise), Tīkṣṇauṣadha (use of sharp drugs), Śoka (grief), Roga (disease), Bhaya (fear) and Upavāsa (fasting) etc., the Vāta gets aggravated in the Mahāsrotas and it vitiates the doṣas and moves them upwards. These doṣas agitating the Āmāśaya (stomach) and compressing the Marma (a vital organ refers to Hṛdaya (heart)) leads to Vātaja Chardi.

- हृत्पार्श्वपीडामुखशोषमूर्धनाभ्यर्तिकासस्वरभेदतोदैः। उद्गारशब्दप्रबलं सफेनं विच्छिन्नकृष्णं तनुकं कषायम् । कृच्छ्रेण चाल्पं महता च वेगेनार्तोऽनिलाच्छर्दयतीह दुःखम् । (Ca.Ci.20/8-9)
- प्रच्छर्दयेत् फेनिलमल्पमल्पं शूलार्दितोऽभ्यर्दितपार्श्वपृष्ठः । श्रान्तः सघोषं बहुशः कषायं जीर्णेऽधिकं साऽनिलजा वमिस्तु ॥ (Su.U.49/9)

The clinical features of Vātaja Chardi are Hṛtpārśvapīḍā (pain in the cardiac region and flanks), Mukha śoṣa (dryness of the mouth), Mūrdhanābhyarti (pain in the head and umbilical region), Kāsa (cough), Svarabheda (hoarseness of voice) and Toda (pricking pain); He vomits with Udgāraśabdaprabalaṃ (very loud eructation), Kṛcchreṇa (with difficulty), Cālpa (in small quantity), Mahatā vega (great force) and Dukha (discomfort); and the vomitus is Saphena (froathy), Vicchinna (scattered), Kṛṣṇa (black in color), Tanu (thin), Kaṣāya (astringent taste or colour or both) and Jīrṇoadhika (gets intensified after digestion).

Pittaja Chardi

- अजीर्णकट्वम्लविदाह्यशीतैरामाशये पित्तमुदीर्णवेगम् । रसायनीभिर्विसृतं प्रपीड्य मर्मोर्ध्वमागम्य वमिं करोति ॥ (Ca.Ci.20/10)

Due to Ajīrṇa (intake of food during indigestion), Kaṭvamla (use of pungent and sour substances), Vidāhya (food which causes burning sensation) and Aśītaīḥ (hot substances), the Pitta in the Āmāśaya (stomach) gets aggravated and spreads through Rasāyani's (Rasa carrying channels) and compressing the Ūrdhvamāgamya Marmās (vital organs in the upper part of the body) and produces Pittaja Chardi.

- मूर्च्छापिपासामुखशोषमूर्धताल्वक्षिसन्तापतमोभ्रमार्तः। पीतं भृशोष्णं हरितं सतिक्तं धूम्रं च पित्तेन वमेत् सदाहम् । (Ca.Ci.20/11)
- योऽम्लं भृशं वा कटुतिक्तवक्त्रः पीतं सरक्तं हरितं वमेद्वा । सदाहचोषज्वरवक्त्रशोषो मूर्च्छान्वितःपित्तनिमित्तजा सा ॥ (Su.U.49/10)

The clinical features of Pittaja Chardi are Mūrcchā (fainting), Pipāsā (thirst), Dāha (burning sensation), Coṣa (sucking sensation), Jvara (fever), Vaktraśoṣa or Mukhaśoṣa (dryness of mouth), Mūrdhatālvakṣisantāpa (heat sensation in the head, palate and eyes), Tamaḥ (feeling of darkness before eyes) and Bhrama (giddiness); and the vomitus is Pīta (yellow), Sarakta (reddish or with blood), Harita (greenish), Bhṛśoṣṇa (with excessive hot), Amla bhṛśa (with excessive sour), Kaṭutiktavaktra or Satikta (pungent and bitter taste in the mouth or bitter taste in the mouth), Dhūmraṃ (smoky in appearance) and Sadāha (with burning sensation)

Kaphaja Chardi

- स्निग्धातिगुर्वामविदाहिभोज्यैः स्वप्नादिभिश्चैव कफोऽतिवृद्धः । उरः शिरो मर्म रसायनीश्च सर्वाः समावृत्य वमिं करोति ॥ (Ca.Ci.20/12)

Due to intake of Snigdha bhojana (unctuous food substances), Atiguru bhojana (very heavy food substances), Āma bhojana (uncooked food substances), Vidāhi bhojyaiḥ (food which causes burning sensation) and Svapnādibhi (excessive sleep etc.), the Kapha gets aggravated and it covers the Uraḥ (chest), Śira (head), Marma (vital organs) and Sarva Rasāyani's (all channels) to produce Kaphaja Chardi.

- तन्द्रास्यमाधुर्यकफप्रसेकसन्तोषनिद्रारुचिगौरवार्तः। स्निग्धं घनं स्वादु कफाद्विशुद्धं सलोमहर्षोऽल्परुजं वमेत्तु ॥ (Ca.Ci.20/13)
- यो हृष्टरोमा मधुरं प्रभूतं शुक्लं हिमं सान्द्रकफानुविद्धम् । अभक्तरुग्गौरवसादयुक्तो वमेद्वमी सा कफकोपजा स्यात् ॥ (Su.U.49/11)

The clinical features of Kaphaja Chardi are Tandra (drowsiness), Āsyamādhurya (sweet taste in the mouth), Kaphapraseka (salivation), Santoṣa (satiation), Nidra (excessive sleep), Aruci (anorexia), Gaurava (heaviness), Lomaharṣa or Hṛṣṭaromā (horripilation), Sāda (malaise) and Alparuja (mild pain); and the vomitus is Snigdha (unctuous), Sāndra or Ghana (thick), Svādu (sweet), Viśuddha (clean) Śukla (white) and Hima (cold).

Sannipātaja Chardi

- समश्नतः सर्वरसान् प्रसक्तमामप्रदोषर्तुविपर्ययैश्च । सर्वे प्रकोपं युगपत् प्रपन्नाश्छर्दिं त्रिदोषां जनयन्ति दोषाः॥ (Ca.Ci.20/14)

Due to Samaśnata (indulging in wholesome and unwholesome food simultaneously), Sarvarasa (use of all taste together), Prasaktamāmapradoṣa (excessive vitiation of Āma doṣa) and Ṛtu viparyaya (contrariety of season), all the three doṣas get aggravated simultaneously and produces Sannipātaja Chardi. Vāgbhaṭa describes that it should be rejected from the treatment.

- शूलाविपाकारुचिदाहतृष्णाश्वासप्रमोहप्रबला प्रसक्तम् । छर्दिस्त्रिदोषाल्लवणाम्लनीलसान्द्रोष्णरक्तं वमतां नृणां स्यात् ॥ (Ca.Ci.20/15)
- सर्वाणि रूपाणि भवन्ति यस्यां सा सर्वदोषप्रभवा मता तु । (Su.U.49/12)

The clinical features of Sannipātaja Chardi are Śūla (colic), Avipāka (indigestion), Aruci (anorexia), Dāha (burning sensation), Tṛṣṇā (thirst), Śvāsa (dyspnoea) and Pramoha (severe delusion); and the vomitus is Lavaṇāmla (salty and sour), Nīla (blue), Sāndra (thick), Uṣṇa (hot) and Rakta (reddish). Suśruta described that all the clinical features of all the three doṣas are present in the Sannipātaja Chardi.

Āgantuja Chardi

- बीभत्सजा दौहृदजाऽऽमजा च सात्म्यप्रकोपात् कृमिजा च या हि ।
  सा पञ्चमी तां च विभावयेत्तु दोषोच्छ्रयेणैव यथोक्तमादौ ॥ (Su.U.49/12)
- बीभत्सजा द्विष्टाशुचिपूत्यमेध्यादिकाद्घृणाकराज्जाता। दौहृदजा दौहृदविमाननाज्जाता, अन्ये दौहृदजां गर्भनिमित्तामाहुः। आमजा विसूच्यादिजाता। सात्म्यप्रकोपात् अकस्मात् सात्म्यत्यागात्। कृमिभिः कृता कृमिजा। (Ḍa.Su.U.49/12)

Suśruta described five types of Āgantuja Chardi namely Bibhatsajā (smell, hearing, sight and thought of substances which are hated, impure, putrid, unholy and disgusting), Dauhṛdajā (pregnancy), Āmaja (Visūcikā etc., Āma pradoṣaja vikāra), Sātmyaprakopaja (sudden withdrawal of wholesome food substances) and Kṛmijā (worm infestations); these should be duly classified according to predominance of doṣas as described above.

Dviṣṭārthayogaja Chardi

- ⊙ द्विष्टप्रतीपाशुचिपूत्यमेध्यबीभत्सगन्धाशनदर्शनैश्च। यच्छर्दयेत्तप्तमना मनोघ्नैर्द्विष्टार्थसंयोगभवा मता सा॥ (Ca.Ci.20/18)

Gandha (smelling), Āśana (eating) and Darśana (seeing) of substances which are Dviṣṭa (hated), Pratipā (Sātmyaviparīta – contrary to wholesome), Aśuci (unclean or impure), Pūtya (putrid), Amedhya (Malina – dirty or unholy) and Bibhatsa (disgusting) inflicts the Manas (psyche) and produces Chardi associated with Hṛdi kliṣṭe (pain in the cardiac region - Vāgbhaṭa); which is called as Dviṣṭārthasaṃyogaja chardi i.e. vomiting due to contact with disliked sensory objects.

Kṛmija Chardi

- ⊙ शूलहृल्लासबहुला कृमिजा च विशेषतः । कृमिहृद्रोगतुल्येन लक्षणेन च लक्षिता । (Su.U.49/13)

The clinical features of Kṛmija Chardi are Śūla (colic), Hṛllāsa bahulā (excessive nausea) and especially Kṛmihṛdroga tulya lakṣaṇās (all the features as in Kṛmija Hrḍroga.

Upadravā

- ⊙ विट्स्वेदमूत्राम्बुवहानि वायुः स्रोतांसि संरुध्य यदोर्ध्वमेति। उत्सन्नदोषस्य समाचितं तं दोषं समुद्धूय नरस्य कोष्ठात्॥ विण्मूत्रयोस्तत् समवर्णगन्धं तृट्श्वासहिक्कार्तियुतं प्रसक्तम्। प्रच्छर्दयेद्दुष्टमिहातिवेगात्तयाऽर्दितश्चाशु विनाशमेति॥ (Ca.Ci.20/16-17)

When the aggravated Vāta obstructing the Viṭsvedamūtrāmbuvaha srotāṃsi (channels carrying faeces, sweat, urine and water) and moves upwards, then the accumulated doṣas from the Koṣṭha (gastrointestinal tract) of the person whose doṣas are aggravated get incited to cause Chardi, which has the following clinical features

Viṇmūtrayostat samavarṇa gandha (vomitus having the colour and smell of the feces and urine) and it contains Duṣṭa (vitiated or putrid materials) and comes with Ativega (strong force) along with Tṛṭ (thirst), Śvāsa (dyspnoea), Hikkā (hiccup) and Arti (discomfort); and such person gets Vināśamāśu (dies quickly).

Sādhyāsādhyata

- ⊙ क्षीणस्य या छर्दिरतिप्रवृद्धा सोपद्रवा शोणितपूययुक्ता। सचन्द्रिकां तां प्रवदन्त्यसाध्यां साध्यां चिकित्सेदनुपद्रवां च॥ (Ca.Ci.20/19)
- ⊙ क्षीणस्योपद्रवैर्युक्तां सासृक्पूयां सचन्द्रिकाम् । छर्दिं प्रसक्तां कुशलो नारभेत चिकित्सितुम् ॥ (Su.U.49/13)

Kṣīṇasya ya chardiratipravṛddhā (vomiting in an emaciated person), Sopadrava (associated with complications) and the vomitus containing Śoṇitapūyayuktā (blood and pus) and Sacandrikā (circular patches with variegated colour), are considered Asādhya (incurable). That which is Sādhya (curable) and Anupradrava (free from complications) should be treated.

In Contemporary science,

Chardi can be predominantly characterised by conditions like vomiting in contemporary science as it is rather seen as a symptom in many diseases than a disease itself. Vomiting is a forceful expulsion of gastric contents as a result of involuntary contraction of the diaphragm; intercostal muscles and abdominal muscles combined with relaxation of lower oesophageal sphincter and it may be acute or chronic. The most common causes of vomiting are alcoholism, peptic ulcer disease, gastroparesis, appendicitis, cholecystitis, pancreatitis, intestinal obstruction, diabetic ketoacidosis, addison's disease, hepatitis, gastroenteritis, urinary tract infection, uraemia, migraine, meningitis, psychogenic and drugs such as NSAIDs, opiates, digoxin, antibiotics and cytotoxins.

Clinical features:

The features of vomiting include retching, abdominal pain, abdominal cramping or tenderness, drowsiness, pale skin, headache, lack of appetite, nausea, extreme thirst, sensitivity to light or sound, dizziness, diarrhoea and fever. Alarm features include signs of hypovolemia, neck stiffness, mental status changes, peritoneal signs, distended and tympanitic abdomen.

Diagnosis:

Diagnosis depends on lab tests, which helps to rule out anaemia, dehydration, inflammation, infection, and liver problems. Urinalysis reveals the signs of dehydration, infection and kidney diseases. Upper GI endoscopy, ultrasound scan, gastric emptying test and antral-duodenal motility may be helpful in chronic vomiting.

MCQ's

1. According to Caraka, Chardi is of .... types

a) 3 b) 4
c) 5 d) 7

2. According to Śārṅgadhara, Chardi is of .... types

a) 3 b) 4
c) 5 d) 7

3. *Āgantuja Chardi is classified in to .... types*
   - a) 3
   - b) 4
   - c) 5
   - d) 7
4. *What are the Doṣas responsible for Chardi*
   - a) Udāna and Vyāna
   - b) Udāna and Samāna
   - c) Udāna and Apāna
   - d) Udāna and Prāṇa
5. *The feature of Chardi Pūrvarūpa include all except*
   - a) Praseka
   - b) Pralāpa
   - c) Hṛdayotkleśa
   - d) Dveṣāśana
6. *Ghṛnaja is a type of*
   - a) Chardi
   - b) Tṛṣṇā
   - c) Śvāsa
   - d) Hikkā
7. मूर्धताल्वक्षिसन्ताप is seen in
   - a) Vātaja Chardi
   - b) Pittaja Chardi
   - c) Kaphaja Chardi
   - d) Sannipātaja Chardi
8. रक्तं वमतां is seen in
   - a) Vātaja Chardi
   - b) Pittaja Chardi
   - c) Kaphaja Chardi
   - d) Sannipātaja Chardi
9. विण्मूत्रयोस्तत् समवर्णगन्धं is seen in
   - a) Pūrvarūpa of Chardi
   - b) Upadrava of Chardi
   - c) Sādhya Lakṣaṇa of Chardi
   - d) Asādhya Lakṣaṇa of Chardi
10. शोणितपूययुक्ता। is seen in
    - a) Pūrvarūpa of Chardi
    - b) Upadrava of Chardi
    - c) Sādhya Lakṣaṇa of Chardi
    - d) Asādhya Lakṣaṇa of Chardi
11. What is vomiting characterized by
    - a) Mild nausea
    - b) Forceful expulsion of gastric contents
    - c) Lightheadedness
    - d) Persistent cough

12. Which of the following is not a common cause of vomiting
    - a) Alcoholism
    - b) Peptic ulcer disease
    - c) Asthma
    - d) Hepatitis
13. Which of the following is an alarm feature of vomiting
    - a) Pale skin
    - b) Nausea
    - c) Neck stiffness
    - d) Lack of appetite

Answer Keys

1	c	2	d	3	c	4	a	5	b	6	a	7	b
8	d	9	b	10	d	11	b	12	c	13	c		

Short Answer Questions

1. Explain Nidāna and Saṃprāpti of Chardi
2. Explain the Chardi bheda and its Lakṣaṇās
3. Explain the Āgantuja Chardi in detail

TOPIC 3

Amlapitta

Introduction

Amlapitta is Pitta pradhāna tridoṣaja vyādhi and is one among the Annavaha srotovikāra caused by Agniduṣṭi and it is characterized by Avipāka, Klama, Utkleśa, Tiktāmla udgāra, Gaurava, Hṛtkaṇṭhadāha and Aruci. Ācārya Caraka had not explained Amlapitta as a separate disease but described as one of the symptoms of Pittaja Grahaṇī. Ācārya Vāgbhaṭa described the disease Amlaka which has the similar symptoms such as Antardāha (Koṣṭha dāha), Hṛdśūla and Udgāra. Hence Amlaka can be taken as a synonym of Amlapitta. Ācārya Kāśyapa explained the Nidāna, Samprāpti, Lakṣaṇās and Cikitsā of Amlapitta in detail. In Basavarajīyam, Amlapitta is explained under Pittaja Nānātmaja vikāra. Amlaka and Śuktaka are synonyms of Amlapitta.

Nirūkti and Paribhāṣā

⊙ अम्लपित्तं चेति अम्लगुणोद्रिक्तं पित्तम्। (Ca.Ca.Ci.15/45-49)

Amlapitta is a condition in which Pitta has an excess of Amla guṇa (sour qualities)

⊙ विदाहाद्यम्लगुणोद्रिक्तं पित्तमम्लपित्तम्। (Ma.M.N.51/1)

⊙ विदग्धं पित्तमम्लपित्तमिति। (Ḍa.Su.Sū.21/11)

⊙ विदाहाद्यम्लगुणोद्रिक्तं पित्तमम्लपित्तमित्युच्यते ॥ (Ād.Śa.P.7/103)

Amlapitta is a condition in which the Pitta becomes Vidagdha (augmented) because of excessive increase of Amla guṇa (sour qualities).

⊙ विदग्धं शुक्ततां याति शुक्तमामाशये स्थितम् । तदम्लपित्तमित्याहुर्भूयिष्ठं पित्तदूषणात् ॥ (Kā.Sa.Khi.16/9)

Due to Vidagdha (improper digestion), the food becomes Śuktatā (sour or acidified) and gets localized in the stomach resulting in Pittadūṣaṇāt (vitiation of Pitta) and produces the disease Amlapitta.

Nidāna and Samprāpti

⊙ विरुद्धाध्यशनाजीर्णादामे चामे च पूरणात्। पिष्टान्नानामपक्वानां मद्यानां गोरसस्य च॥
गुर्वविष्यन्दिभोज्यानां वेगानां धारणस्य च। अत्युष्णस्निग्धरूक्षाम्लद्रवाणामतिसेवनात्॥
फाणितेक्षुविकाराणां कुलत्थानां च शीलनात्। भृष्टधान्यपुलाकानां पृथुकानां तथैव च॥
भुक्त्वा भुक्त्वा दिवास्वप्नादतिस्नानावगाहनात्। अन्तरोदकपानाच्च भुक्तपर्युषिताशनात्॥
वातादयः प्रकुप्यन्ति तेषामन्यतमो यदा। मन्दीकरोति कायाग्निमग्नौ मार्दवमागते॥
एतान्येव तथा भूयः सेवमानस्य दुर्मतेः। यत्किञ्चिदशितं पीतं देहिनस्तद्धि दह्यति॥
विदग्धं शुक्ततां याति शुक्तमामाशये स्थितम्। तदम्लपित्तमित्याहुर्भूयिष्ठं पित्तदूषणात्॥
जन्तोर्यदनुबध्नाति लौल्यादनियतात्मनः॥ (Kā.Sa.Khi.16/3-10)

Due to intake of Viruddhāśana (contrary food substances), Adhyaśana (eating before the digestion of previous meal), Āma (presence of Āma), Āme pūraṇāt (filling of body with Āma), Piṣṭānna (food prepared from flour), Apakvamadya (uncooked wine), Apakva gorasa (uncooked milk), Guru bhojana (heavy food substances), Abhiṣyandi bhojana (moisture producing foods), Vegadhāraṇa (suppressing natural urges), Atyuṣṇa snigdha rūkṣāmla dravaṇāmatisevanāt (excessive intake of very hot, unctuous, dry, sour and liquid substances), intake of Phāṇitekṣu vikārāṇāṃ (unfermented sugarcane products such as molasses), Kulattha, Bhṛṣṭadhānya (parched cereals), Pulākānāṃ (lump of boiled rice), Pṛthukānāṃ (flattened rice), Bhuktvā bhuktvā divāsvapna (day sleeping after eating again and again), Atisnānāt (excessive bathing), Avagāhanāt (tub bath), Antarodaka pānācca bhukta (drinking water in between food) and Paryuṣitāśanāt (stale food) aggravation of Vātādi doṣas takes place resulting in Kāyāgnimandyata (diminished digestive fire). In this stage, whatever is eaten or drunk by ignorant person, the same gets digested. Due to Vidagdha (improper digestion), the food becomes Śuktatā (sourness or acidified) and gets localized in the stomach and also when the person who is Aniyatātmanaḥ (not having self control) eats due to Laulya (greed), it leads to Pittadūṣaṇāt (vitiation of Pitta) and produces the disease Amlapitta.

⊙ विरुद्धदुष्टाम्लविदाहिपित्तप्रकोपिपानान्नभुजो विदग्धम् । पित्तं स्वहेतूपचितं पुरा यत्तदम्लपित्तं प्रवदन्ति सन्तः । (M.N.51/1) / (Y.R.57/1)

Due to intake of food which are Viruddha (contrary), Duṣṭa (contaminated or spoiled), Amla (very sour), Vidāhi (causing burning sensation) or Pittaprakopipānna bhujo (intake food and drinks causing increase of Pitta) by the person already having accumulation of Pitta due to its own factors, the aggravated Pitta gets Vidagdhaṃ (improperly burnt) and produces the disease Amlapitta.

Samprāpti Ghaṭaka

Doṣa	Pitta Pradhāna Tridoṣas
Dūṣya	Rasa
Agni	Jaṭharāgni māndhya

Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa and Anna vaha
Srotoduṣṭi	Saṅga and Vimārga gamana
Udbhava sthāna	Āmāśaya
Sañcāra sthāna	Mahā srotas
Vyakta sthāna	Āmāśaya
Adhiṣṭhāna	Āmāśaya
Rogamārga	Abhyantara

Sāmānya Lakṣaṇās

- अविपाकक्लमोत्क्लेशतिक्ताम्लोद्गारगौरवैः । हृत्कण्ठदाहारुचिभिश्चाम्लपित्तं वदेद्भिषक् ।
  (M.N.51/2) / (Y.R.57/2)

Avipāka (indigestion), Klama (exhaustion), Utkleśa (nausea), Tiktāmla udgāra (bitter and sour eructation), Gaurava (heaviness of the body), Hṛtkaṇṭhadāha (burning sensation in the cardiac and throat region) and Aruci (anorexia) are the general clinical features of Amlapitta.

- विड्भेदो गुरुकोष्ठत्वमम्लोत्क्लेशः शिरोरुजा । हृच्छूलमुदराध्मानमङ्गसादोऽन्त्रकूजनम् ॥
  कण्ठोरसी विदह्येते रोमहर्षश्च जायते। सामान्यलक्षणं त्वेत.... ॥ (Kā.Sa.Khi.16/14-15)

Viḍbheda (diarhhoea), Gurukoṣṭha (heaviness of abdomen), Amlotkleśa (sour eructation), Śirorujā (headache), Hṛcchūla (pain in the cardiac region), Udarādhmāna (distension of abdomen), Aṅgasāda (lethargy), Antrakūjana (gurgling sound in the abdomen), Kaṇṭhorasī vidahyete (burning sensation in the throat and chest) and Romaharṣa (horripilation) are the general clinical features of Amlapitta.

Bheda and Lakṣaṇās

Ācārya Kāśyapa classified Amlapitta into three types based on the predominance of doṣas i.e. Vātaja, Pittaja and Kaphaja.

- वाताच्छूलाङ्गसादो च जृम्भा स्निग्धोपशायिता। पित्ताद्भ्रमो विदाहश्च स्वादुशीतोपशायिता॥
  कफाद् गुरुत्वं छर्दिश्च स्याद्रूक्षोष्णोपशायिता॥ (Kā.Sa.Khi.16/16-17)
  - Śūla (Pricking pain), Aṅgasāda (lethargy) and Jṛmbhā (yawning) are the features of Vātaja Amlapitta and Snigdhopaśāyitā (unctuous substances is indicated for allaying the disease).

  - Bhrama (giddiness) and Vidāha (burning sensation) are the features of Pittaja Amlapitta and Svāduśītopaśāyitā (sweet and cold substances are indicated for allaying the disease).
  - Gurutva (heaviness) and Chardi (vomiting) are the features of Kaphaja Amlapitta and Rūkṣoṣṇopaśāyitā (non-unctuous and hot substances are indicated for allaying the disease).

Ācārya Mādhavakara classified Amlapitta into two types based on the Pravṛtti (manifestation) i.e. Ūrdhvaga and Adhoga Amlapitta.

Adhoga Amlapitta

- तृड्दाहमूर्छाभ्रममोहकारि प्रयात्यधो वा विविधप्रकारम् । हृल्लासकोठानलसादहर्षस्वेदाङ्ग पीतत्वकरं कदाचित् ॥
  (M.N.51/3) / (Y.R.57/3)

The features of Adhoga Amlapitta are Tṛḍ (thirst), Dāha (burning sensation), Mūrcchā (fainting), Bhrama (giddiness), Moha (delusion) and Prayātyadho vā vividhaprakāra (passes different types of stool downwards), hence this condition is called Adhoga Amlapitta. Sometimes, there will be Hṛllāsa (nausea), Koṭha (urticaria), Analasāda (diminution of digestive fire), Harṣa (horripilation), Sveda (perspiration) and Aṅgapītatvakaraṃ (yellowish complexion of the body).

Ūrdhvaga Amlapitta

- वान्तं हरित्पीतकनीलकृष्णमारक्तरक्ताभमतीव चाम्लम् । मांसोदकाभं त्वतिपिच्छिलाच्छं श्लेष्मानुजातं विविधं रसेन॥
  भुक्ते विदग्धे त्वथवाऽप्यभुक्ते करोति तिक्ताम्लवमिं कदाचित् । उद्गारमेवंविधमेव कण्ठहृत्कुक्षिदाहं शिरसो रुजं च॥
  करचरणदाहमौष्ण्यं महतीमरुचिं ज्वरं च कफपित्तम् । जनयति कण्डूमण्डलपिडकाशतनिचितगात्ररोगचयम् ॥
  (M.N.51/4-6) / (Y.R.57/4-6)

The features of Ūrdhvaga Amlapitta are Vāntaṃ haritpītakanīlakṛṣṇamārakta raktābham (vomitus is green, yellow, blue, black, blood mixed or blood like), Atīva cāmlam (vomitus has excessive sourness), Māṃsodakābhaṃ (resembling the meat washed water), Atipicchilā (excessive sliminess), Acchaṃ (transparent) and Vividha rasa (varied taste) along with Kapha. Bhukte vidagdheapyathavāpyabhukte karoti tiktāmla vamiṃ (sometimes vomiting occurs during digestion of food or in empty stomach with bitter or sour taste), sometimes there will be Tiktāmla udgāra (bitter or sour eructation), Kaṇṭhahṛtkukṣidāha (burning sensation in the throat, cardiac region and abdomen), Śiraso rujaṃ (headache), Karacaraṇadāhamauṣṇyaṃ (burning sensation and warmth in the hands and feet), Mahatīmaruci (severe anorexia), Kaphapittaja Jvara, Kaṇḍū (itching sensation), Maṇḍala (circular patches), Piṭika (eruption of furuncles) and Ācitagātrarogacayam (other various diseases) are inflicted to the body.

- सानिलं सानिलकफं सकफं तच्च लक्षयेत् । दोषलिङ्गेन मतिमान् भिषङ्मोहकरं हि तत् ॥ (M.N.51/8)

Again Ācārya Mādhavakara classified Amlapitta into four types based on the doṣānubandha i.e. Sānila, Sānilakapha, Sakapha and Śleṣmapitta

Sānila Amlapitta

⦿ कम्पप्रलापमूर्छाचिमिचिमिगात्रावसादशूलानि । तमसो दर्शनविभ्रमविमोहहर्षाण्यनिलकोपात् ॥ (M.N.51/9)

⦿ कम्पप्रलापमूर्च्छाचिमिचिमिगात्रावसादशूलानि । तमसो दर्शनविभ्रमप्रमोहहर्षा अनिलयुते ॥ (Y.R.57/9)

Kampa (tremors), Pralāpa (delirium), Mūrchhā (fainting), Cimicimi (tingling sensation), Gātrāvasāda (malaise), Śūla (pain), Tamasodarśana (darkness before eyes), Vibhrama (giddiness), Moha (delusion) and Harṣa (horripilation) are the symptoms caused by Vātānubandha Amlapitta

Sakapha Amlapitta

⦿ कफनिष्ठीवनगौरवजडतारुचिशीतसादवमिलेपाः । दहनबलसादकण्डूनिद्राश्चिह्नं कफानुगते ॥ (M.N.51/10) / (Y.R.57/10)

Kapha niṣṭhivana (expectoration of thick phlegm), Gaurava (heaviness), Jaḍata (stiffness), Aruci (anorexia), Śīta (coldness), Sāda (malaise), Vami (vomiting), Lepā (coating on the tongue), Dahana (burning sensation), Balasāda (loss of strength), Kaṇḍū (itching sensation) and Nidrā (sleepiness) are the symptoms caused by Kaphānubandha Amlapitta.

Sānilakapha Amlapitta

⦿ उभयमिदमेव चिह्नं मारुतकफसम्भवे भवत्यम्ले । (M.N.51/11) / (Y.R.57/11)

Appearances of aforesaid features are seen together in Amlapitta in which both Vāta and Kapha are associated.

Śleṣmapitta Amlapitta

⦿ तिक्ताम्लकटुकोद्गारहृत्कुक्षिकण्ठदाहकृत् ॥ भ्रमो मूर्च्छाऽरुचिश्छर्दिरालस्यं च शिरोरुजा ।
प्रसेको मुखमाधुर्यं श्लेष्मपित्तस्य लक्षणम् ॥ (M.N.51/11-12)

⦿ कट्वम्ललवणरसासेविते कोपं भजत्येवा तिक्ताम्लकटुकोद्गारवमिहृत्कण्ठदाहकृत् ॥
भ्रमो मूर्च्छाऽरुचिश्छर्दिरालस्यं च शिरोरुजा ॥ प्रसेको मुखमाधुर्य श्लेष्मपित्तस्य लक्षणम् ॥ (Y.R.57/11-12)

Due to excessive intake of Kaṭvamlalavaṇarasasevite (bitter, sour and saline substances) causes aggravation of Śleṣmapitta doṣa and resulting in Tiktāmlakaṭukodgāra (bitter, sour and pungent eructation), Vami (vomiting), Hṛtkaṇṭhadāha (burning sensation in the cardiac and throat region), Bhrama (giddiness), Mūrcchā (fainting), Aruci (anorexia), Chardi (vomiting), Ālasya (languor), Śirorujā (headache), Praseka (salivation) and Mukhamādhurya (sweet taste in the mouth).

Upadrava

⦿ ज्वरातीसारपाण्डुत्वशूलशोथारुचिभ्रमैः । उपद्रवैरिमैर्जुष्टः क्षीणधातुर्न सिद्ध्यति ॥ (Kā.Sa.Khi.16/49)

Jvara (fever), Atisāra (diarhhoea), Pāṇḍutva (anaemia), Śūla (colic), Śotha (oedema), Aruci (anorexia) and Bhrama (giddiness) are the complications of Amlapitta and also Kṣīṇadhātu (loss of dhātus), which does not get cured.

Sādhyāsādhyata

⦿ रोगोऽयमम्लपित्ताख्यो यत्नात् संसाध्यते नवः । चिरोत्थितो भवेद्याप्यः, कृच्छ्रसाध्यः स कस्यचित् ॥
(M.N.51/7) / (Y.R.57/7)

The disease Amlapitta which is of Nava (recent onset) is Yatnātsaṃsādhya (curable with effort), that which is of Cirotthita (chronic one) is Yāpya (palliable) and sometimes can become Kaṣṭasādhya (difficult to cure).

In Contemporary science,

Amlapitta can be predominantly characterised by conditions like acid peptic disorders in contemporary science. Acid peptic disorders (APD) occurs as a result of distinctive, but overlapping pathogenic mechanisms leading to either excessive acid secretion or diminished mucosal defense, which mainly includes Gastro-esophageal reflux disease (GERD) and Peptic ulcer disease (PUD). The prevalence of GERD in India is likely to be between 8% and 19% in general population and another epidemiology study reported a prevalence of 7.8% for PUD in India.

Gastro-esophageal reflux disease (GERD)

Gastro-esophageal reflux disease (GERD) is the most common disorder of the oesophagus. Gastro-esophageal reflux disease (GERD) is incompetence of the lower oesophageal sphincter which allows reflux of gastric contents into the oesophagus, causing burning pain. It is known as reflux oesophagitis if there is damage to the oesophageal epithelium. The main causative factor of GERD is transient lower oesophageal sphincter relaxation. Obesity, hiatus hernia, pregnancy, connective tissue disorders such as scleroderma, delayed stomach emptying, smoking, eating large meals or eating late at night, eating fatty or fried foods, alcohol, coffee, drugs like aspirin are the factors increasing the risk of GERD.

Clinical features: Heart burn and regurgitation of sour taste in the mouth are the common symptoms. Other symptoms include chest pain, persistent dry cough, hoarseness or sore throat, asthma, dental erosions and globus sensation (feeling of fullness or a lump in the back of throat). Alarm symptoms include dysphagia, odynophagia (painful swallowing), weight loss, anaemia and bleeding.

Complications: Barrett's oesophagus, peptic oesophageal ulcers and benign oesophageal strictures and malignancy.

Diagnosis: Diagnosis is based on the clinical presentation, endoscopy and pH metry. GERD is usually diagnosed clinically with classic symptoms i.e. heartburn and regurgitation.

Endoscopy helps to visualize the oesophageal mucosa and its possible complications such as strictures, ulcers, Barrett's esophagus and oesophageal adeno-carcinoma. Ambulatory pH monitoring for 24 hours helps in the evaluation of acid reflux.

MCQ's

1. Synonyms of Amlapitta
   a) Amlaka b) Śuktaka
   c) Both a and b d) None of the above
2. Ācārya Kāśyapa classified Amlapitta in to
   a) Vātaja, Pittaja and Kaphaja
   b) Adhoga and Ūrdhvaga
   c) Sānila, Sānilakapha, Sakapha and Śleṣmapitta
   d) None of the above
3. Amlapitta is of .... types, based on the Pravṛtti
   a) 2 b) 3
   c) 4 d) 5
4. Amlapitta is of .... types, based on the doṣa predominance
   a) 2 b) 3
   c) 4 d) 5
5. तिक्ताम्लोद्गार is seen in
   a) Jvara b) Raktapitta
   c) Āmavāta d) Amlapitta
6. अङ्ग पीतत्वकरं is seen in
   a) Adhoga Amlapitta b) Ūrdhvaga Amlapitta
   c) Sānila Amlapitta d) Sakapha Amlapitta
7. महतीमरुचिं is seen in
   a) Adhoga Amlapitta b) Ūrdhvaga Amlapitta
   c) Sānila Amlapitta d) Sakapha Amlapitta

8. The feature of Amlapitta Upadrava include all except
   a) Jvara b) Atisāra
   c) Pāṇḍu d) Kāmalā
9. What is reflux oesophagitis?
   a) Inflammation of the oesophagus due to infection
   b) Damage to the oesophageal epithelium caused by reflux
   c) Obstruction of the oesophagus by a foreign body
   d) Spasm of the oesophageal muscles
10. Which of the following is a common symptom of GERD?
   a) Chronic cough b) Persistent rash
   c) Joint pain d) Severe headache

Answer Keys

1	c	2	a	3	a	4	c	5	d
6	a	7	b	8	d	9	b	10	a

Short Answer Questions

1. Explain Nidāna and Samprāpti of Amlapitta
2. Explain Adhoga Amlapitta
3. Explain Ūrdhvaga Amlapitta

◆

TOPIC 4

Śūla Roga

Introduction

Śūla roga means colic which occurs at the site of Gulma without its presence (Vinā gulmena yacchūlaṃ) and it may occur as a separate disease or as a symptom in other diseases. Śūla occurs as a complication of Gulma; hence Ācārya Suśruta explained Śūla along with Gulma. Ācārya Vāgbhaṭa while describing the Raktaja Gulma stated that the Śūla occurs as an earliest symptom and the Gulma manifests later; whereas Ācārya Caraka stated that the Śūla occurs in the Gulma as a pathological event. But both the Ācāryās have not dedicated a separate chapter for Śūla.

Ācārya Hārita described a mythological story of Śūla that Lord Shiva (Hara) once got angry on Anaṅga (Kāmadeva) and he threw his Triśūla (trident) to kill Kāmadeva, on seeing the dreadful trident approaching him, frightened Makaradhvaja (Kāmadeva) suddenly entered the body of Lord Viṣṇu; and Lord Viṣṇu produced the Huṅkāra (menacing Huṃ sound), by that sound the trident being confused and fell on the Bhūmi (earth), that is known as Śūla. And it afflicts the Pañcabhūtanugata śarīra of the people and absorbs the energy and produced Śūla roga.

Nirūkti and Paribhāṣā

- ⊙ शूल रुजायाम् । शूलति लोकं रोगः । (SKD)
- ⊙ शूलति लोकानिति शूल रोगे । (SKD)

The term Śūla means Rujāyām (be sick or ill) and it is called as Loka rogaḥ (disease of mankind).

- ⊙ शूलनिर्घातवेदनाजनकत्वाच्छूलसंज्ञा ॥ (Āḍ.Śā.P.7/42)

That which produces Nirghāta Vedana (destructive type of pain) is known as Śūla.

- ⊙ शूलं निखानितमिवासुखं येन तु वेत्त्यसौ । (Su.U.42/67)
- ⊙ शूलं निखानितमिव कीलकं निखानितमिवेत्यर्थः। (Ḍa.Su.U.42/67)

That which produces Asukha (discomfort) like that of Kīlaka nikhānita (as if being pricked by pin or spike) is known as Śūla.

- ⊙ शूलसम्भवत्वादस्य शूलमिति सञ्ज्ञा, शूलनिखातवद्वेदनाजनकत्वाच्च। (Ma.M.N.26/1)

The word Śūla refers to spike – a weapon. The discomfort or pain felt which is similar to Śūla sambhavatvāt (being pierced by spike) or Nikhātavad (being digging in or thrust off) is called Śūla.

- ⊙ शूलरोपणवत्पीडा यस्याकस्मात्प्रजायते । त्रिशूलसंभवं चैनं शूलमाहुः पुराविदः॥ (Y.R.29/3)

That which produces Ropaṇavatpīḍa (severe pain as if being pierced by an arrow) is known as Śūla according to ancient scholars because it appeared first due to the Triśūla (trident).

- ⊙ शङ्कुस्फोटनवत्तस्य यस्मात्तीव्राश्च वेदनाः । शूलासक्तस्य लक्ष्यन्ते तस्माच्छूलमिहोच्यते ॥ (Su.U.42/81)
- ⊙ शङ्कस्फोटनवत्तस्य यस्मात्तीव्र च वेदना । शूलासक्तस्य भवति तस्माच्छूलमिहोच्यते ॥ (Y.R.29/4)

That which produces the Tīvrā vedanā (severe pain) and Śūlāsaktasya (excruciating pain) as if being pierced by a Śaṅku (spear or nail) is known as Śūla.

- ⊙ यतश्च तस्मिन् शूलविद्ध इव व्यथते तीव्रवेदनार्दितः कृच्छ्रोच्छ्वासः तस्माच्छूलमित्युच्यते॥ (A.S.Ni.11/32)
- ⊙ गुल्मो नाम्ना शूलमित्युच्यते। (In.A.S.Ni.11/32)

That which produces Śulaviddha iva vyathate (pain as if pierced by a spear) associated with Tīvravedanā (excruciating pain) and Kṛcchrocchvāsa (difficulty in breathing) is known as Śūla. Ācārya Indu described that the Gulma is Śūla by name.

Sthāna of Śūla

- ⊙ विना गुल्मेन यच्छूलं गुल्मस्थानेषु जायते ॥ (Su.U.42/76)

Śūla roga means colic which occurs at the site of Gulma without its presence.

- ⊙ वातात्मकं बस्तिगतं वदन्ति पित्तात्मकं चापि वदन्ति नाभ्याम् । हृत्पार्श्वकुक्षौ कफसन्निविष्टं सर्वेषु देशेषु च सन्निपाताद्॥ (Y.R.29/18)

Vātaja Śūla occurs in Basti (urinary bladder or pelvic region), Pittaja Śūla occurs in Nābhi (umbilical region), Kaphaja Śūla occurs in Hṛdi (cardiac region) Pārśva (both flanks) and Kukṣau (abdomen) and Sannipātaja Śūla occurs in Sarva sthāna (all the above said locations).

Sāmānya Nidāna and Saṃprāpti

- ⊙ वातमूत्रपुरीषाणां निग्रहादतिभोजनात् ॥ अजीर्णाध्यशनायासविरुद्धान्नोपसेवनात् ।

पानीयपानात् क्षुत्काले विरूढानां च सेवनात् ॥ पिष्टान्नशुष्कमांसानामुपयोगात्तथैव च ।
एवंविधानां द्रव्याणामन्येषां चोपसेवनात् ॥ वायुः प्रकुपितः कोष्ठे शूलं सञ्जनयेद्भृशम् ।
निरुच्छ्वासी भवेत्तेन वेदनापीडितो नरः ॥ (Su.U.42/77-80)

Vātamūtrapurīṣāṇāṃ nigraha (suppressing the flatus, urine and faeces), Atibhojanāt (excessive intake of food), Ajīrṇā (indigestion), Adhyaśana (eating before the digestion of previous food), Āyāsa (exertion), Viruddhānnopasevanāt (intake of incompatible food), Pānīyapānāt kṣutkāle (drinking fluid when hungry), Virūḍhanāṃ sevanāt (intake of germinated grains), Piṣṭānnaśuṣkamāṃsa upayogāt (use of cake and pastries and dry meat) and Evaṃvidhānāṃ dravyāṇamanyeṣāṃ upasevanāt (intake of other substances in the similar way) cause the Vitiation of Vāta resulting in Bhṛśa koṣṭha śūla (severe abdominal pain) and it is associated with Nirucchvāsī (breathlessness).

Saṃprāpti Ghaṭaka

Doṣa	Vāta Pradhāna Tridoṣas
Dūṣya	Rasa
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa and Anna vaha
Srotoduṣṭi	Saṅga
Udbhava sthāna	Āmapakvāśaya
Sañcāra sthāna	Koṣṭha (Mahā srotas)
Vyakta sthāna	Gulma sthāna i.e. (Pañcasthānās - Hṛdaya, Nābhi, Basti and Pārśva)
Adhiṣṭhāna	Koṣṭha (Mahā srotas)
Rogamārga	Abhyantara

heda

Ācārya Suśruta classified Śūla into four types based on the predominance of doṣas namely itaja, Pittaja, Kaphaja and Sannipātaja. And also he described some more types based on the hāna (location) namely, Pārśvaśūla, Kukṣi śūla, Hṛtśūla, Bastiśūla, Mūtraśūla, Purīṣa śūla d Avipākādbhavecchūla (Annaja Śūla).

- पृथग्दोषैः समस्तामद्वन्द्वैः शूलोऽष्टधा भवेत् । सर्वेष्वेतेषु शूलेषु प्रायेण पवनः प्रभुः॥ (Y.R.29/5)

Yogaratnākara and Mādhavakara classified Śūla into eight types namely Vātaja, Pittaja, Kaphaja, Sannipātaja, Āmaja and Dvandaja (Vātapittaja, Pittakaphaja and Kaphavātaja). Vāta is predominant in all types of Śūla. Again Mādhavakara added two more types of Śūla, namely Pariṇāma śūla and Annadrava śūla.

Viśiṣṭa Nidāna and Lakṣaṇās

Vātaja Śūla Nidāna and Lakṣaṇās

- व्यायामयानादतिमैथुनाच्च प्रजागराच्छीतजलातिपानात्। कलायमुद्गाढकिकोरदूषादत्यर्थरूक्षाध्यशनाभिघातात्॥ कषाय तिक्तातिविरूढजान्नविरुद्ध वल्लूरकशुष्कशाकात्। विट्शुक्र मूत्रानिलवेगरोधाच्छोकोपवासादतिहास्यभाष्यात्। वायुः प्रवृद्धो जनयेद्धि शूलं हृत्पार्श्वपृष्ठत्रिकवस्तिदेशे॥ (Y.R.29/6-7)

Excessive indulging in Vyāyāmayānādatimaithunā (exercise, riding and sexual intercourse), Prajāgarana (awakening in the night), Śītajalātipānāt (excessive drinking of cold water), Kalāyamudgāḍhakikoradūṣā (intake of Kalāya, Mudga, Āḍhakī and Koradūṣa), Atyartharūkṣa (excessive intake of dry food), Adhyaśana (eating before the previous meal is digested), Abhighātāt (injury), Kaṣāya tiktāti (excessive intake of astringent and pungent substances), Ativirūḍhajānna (excessive intake of germinated grains), Viruddhānna (intake of incompatible food substances), Vallūrakaśuṣkaśākāt (intake of dry meat and dry vegetables), Viṭśukramūtrānilavegarodha (suppressing the natural urges like faeces, semen, urine and flatus), Śoka (grief), Upavāsa (fasting) and Atihāsyabhāṣyāt (excessive laughing and talking) lead to aggravation of Vāta and produces Hṛtpārśvapṛṣṭhatrikabasti śūla (pain in the cardiac, flanks, back, sacral, and bladder regions).

- जीर्णे प्रदोषे च घनागमे च शीते च कोपं समुपैति गाढम्। मुहुर्मुहुश्चोपशमप्रकोपौ विड्वातसंस्तम्भनतोदभेदैः॥ संस्वेदनाभ्यञ्जनमर्दनाद्यैः स्निग्धोष्णभोज्यैश्च शमं प्रयाति॥ (Y.R.29/8)

The aggravation of Vāta doṣa occurs during Jīrṇe (after digestion of food), Pradoṣāgama (nightfall), Ghanāgame (rainy season or weather) and Śīte (cold). Muhurmuhuścopaśama prakopa (frequently the pain subsides and aggravates), Viṭvātasaṃsthambhana (obstruction of faeces and flatus), Toda (pricking pain), Bheda (breaking pain) are the clinical features of Vātaja Śūla and it is pacified by Saṃsvedana (sudation), Abhyañjana (anointing), Mardanā (massage) etc., and Snigdhoṣṇabhojana (unctuous, and hot food substances).

- निराहारस्य यस्यैव तीव्रं शूलमुदीर्यते । प्रस्तब्धगात्रो भवति कृच्छ्रेणोच्छ्वसितीव च ॥ वातमूत्रपुरीषाणि कृच्छ्रेण कुरुते नरः । एतैर्लिङ्गैर्विजानीयाच्छूलं वातसमुद्भवम् ॥ (Su.U.42/82-83)

The person gets Tīvraśūla (severe abdominal pain) during Nirāhārasya (on empty stomach) and develops Stabdhagātra (stiffening of the whole body), Kṛcchreṇocchvāsa (difficulty in breathing) and Vātamūtrapurīṣāṇi kṛcchreṇa (difficulty in passing the flatus, urine, and faeces) as the clinical features in Vātaja Śūla.

Pittaja Śūla Nidāna and Lakṣaṇās

- ⊙ क्षारातितीक्ष्णोष्णविदाहितैलनिष्पावपिण्याककुलत्थयूषैः। कट्वम्लसौवीरसुराविकारैः क्रोधानलायासरविप्रतापैः॥ ग्राम्यातियोगादशनैर्विदग्धैः पित्तं प्रकुप्याऽऽशु करोति शूलम् । तृण्मोहदाहार्तिकरं हि नाभ्यां संस्वेदमूर्च्छाभ्रमशोषयुक्तम्॥ मध्यन्दिने कुप्यति चार्धरात्रे निदाघकाले जलदात्यये च। शीते च शीतैः समुपैति शान्तिं सुस्वादुशीतैरपि भोजनैश्च॥ (Y.R.29/9-11)

Excessive indulging in Kṣārātitīkṣṇoṣṇa (alkaline, sharp, and hot substances), Vidāhi (causing burning sensation during digestion), Taila (oil), Niṣpāva, Piṇyāka (oil cake), Kulatthayūṣa (soup of Kulattha), Kaṭvamlasauvīrasurāvikāraiḥ (intake of bitter, sour substances, Sauvīra, wine and their preparations), Krodha (anger), Analāyāsaravipratāpaiḥ (exposure to the heat of fire, exhaustion, heat of sun), Grāmyātiyogād (excessive sexual intercourse) and Aśanaīrvidagdhaiḥ (improper digestion of food) lead to aggravation of Pitta and produces Śūla with Tṛṇ (thirst), Moha (delusion), Dāha (burning sensation), Nābhyāṃ artikara (pain in the umbilical region), Saṃsveda (perspiration), Mūrcchā (fainting), Bhrama (giddiness) and Śoṣa (feeling of dryness). Madyandine kupyati cārdharātre nidāghakāle jaladātyaye (the pain aggravates during afternoon, midnight, summer and autumn season) and Śīte ca śītaīh samupaiti śāntiṃ susvāduśiṭairapi bhojana (it relieves by cold therapies and intake of sweet and cold food substances).

- ⊙ तृष्णा दाहो मदो मूर्च्छा तीव्रं शूलं तथैव च । शीताभिकामो भवति शीतेनैव प्रशाम्यति ॥ एतैर्लिङ्गैर्विजानीयाच्छूलं पित्तसमुद्भवम् ॥ (Su.U.42/84-85)

Tṛṣṇā (thirst), Dāha (burning sensation), Mada (intoxication), Mūrcchā (fainting), Tīvraśūla (severe pain), Śītābhikāmo (desire for cold substances) and Śītenaiva praśāmyati (gets relieved by cold therapies) are the clinical features of Pittaja Śūla.

Kaphaja Śūla Nidāna and Lakṣaṇās

- ⊙ आनूपवारिजकिलाटपयोविकारैर्मांसेक्षुपिष्टकृशरातिलशष्कुलीभिः। अन्यैर्बलासजनवैâरपि हेतुभिश्च श्लेष्मा प्रकोपमुपगम्य करोति शूलम्॥ (Y.R.29/12)

Excessive intake of Ānūpavārija māṃsa (meat of marshy or aquatic animals), Kilāṭa (inspissated milk), Payasavikārai (milk and its preparations), Ikṣu (sugarcane), Kṛśarā (dish consisting of sesamum and grain), Tila (sesame), Śaṣkulībhiḥ (rice-gruel or barley-water), Anyairbalāsajanakairapi hetubhiśca (other causes of aggravation of Kapha) leads to Kaphaja Śūla.

- ⊙ हल्लासकाससदनारुचिसम्प्रसेवैâरामाशये स्तिमितकोष्ठशिरोगुरुत्वैः। भुक्ते सदैव हि रुजं कुरुतेऽतिमात्रं सूर्योदयेऽथ शिशिरे कुसुमागमे च॥ (Y.R.29/13)

Hṛllāsa (nausea), Kāsa (cough), Sadana (malaise), Aruci (anorexia), Sampraseka (salivation), Āmāśayestimita (feeling as if stomach is filled with moisture), Koṣṭhaśirogurutva (heaviness of abdomen and head), Bhukte sadaiva hi rujaṃ kuruteatimātraṃ sūryodayeatha śiśire kusumāgame (pain aggravates after intake of food and during sunrise, winter and spring seasons) are the clinical features of Kaphaja Śūla.

- ⊙ शूलेनोत्पीड्यमानस्य हल्लास उपजायते ॥ अतीव पूर्णकोष्ठत्वं तथैव गुरुगात्रता । एतच्छ्लेष्मसमुत्थस्य शूलस्योक्तं निदर्शनम् ॥ (Su.U.42/85-86)

Śūlenotpīdyamānasya Hṛllāsa (feeling of nausea due to abdominal pain), Atīva pūrṇakoṣṭha (excessive fullness of abdomen) and Gurugātratā (heaviness of abdomen) are the clinical features of Kaphaja Śūla.

Sannipātaja Śūla Nidāna and Lakṣaṇās

- ⊙ सर्वेषु दोषेषु च सर्वलिङ्गं विद्याद्भिषव्âं सर्वभवं हि शूलम्। सुकष्टमेनं विषवङ्कातुल्यं विवर्जनीयं प्रवदन्ति तज्ज्ञाः॥ (Y.R.29/14)

In case of Sannipātaja śūla, clinical features of all the three doṣas occur simultaneously and it is troublesome like a Viṣa (poison) or Vajra (Lord Indra's weapon). So, the physician should discard the patient from the treatment.

- ⊙ सर्वाणि दृष्ट्वा रूपाणि निर्दिशेत्सान्निपातिकम् । सन्निपातसमुत्थानमसाध्यं तं विनिर्दिशेत् ॥ (Su.U.42/87)

In case of Sannipātaja śūla, clinical features of all the three doṣas occurs simultaneously and it is Āsādhya (incurable), because it originates from combined vitiation of all the three doṣas.

Āmaja Śūla Nidāna and Lakṣaṇās

- ⊙ आटोपहल्लासवमीगुरुत्वस्तैमित्यकानाहकफप्रसेवैâः। कफस्य लिङ्गेन समानलिङ्गमामोद्भवं शूलमुदाहरन्ति॥ (Y.R.29/15)

The clinical features of Āmaja śūla are Āṭopa (gurgling sound in the abdomen), Hṛllāsa (nausea), Vamī (vomiting), Gurutva (heaviness), Staimitya (feeling as if covered with wet cloth), Ānāha (distension of abdomen and constipation), Kaphapraseka (mucoid salivation) and the features of Kaphaja śūla.

Dvandvaja Śūla Nidāna and Lakṣaṇās

- ⊙ द्विदोषलक्षणैरेतैर्विद्याच्छूलं द्विदोषजम्। वस्तौ हृत्कष्ठापार्श्वेषु स शूलः कफवातिकः॥ कुक्षौ हृन्नाभिमध्ये यः स शूलः कफपैत्तिकः। दाहज्वरकरो घोरो विज्ञेयो वातपैत्तिकः॥ (Y.R.29/16-17)

In case of Dvandaja śūla, clinical features of two doṣas are manifested together. In case of Kaphavātaja śūla, there will be Bastauhṛtkaṇṭhapārśva śūla (pain in the bladder, cardiac region, throat and flanks); in case of Kaphapittaja śūla, there will be Kukṣauhṛnnābhimadhye śūla (pain in the stomach and in between the cardiac and umbilical region) and in case of Vātapittaja śūla, there will be Ghora Dāhajvara (severe burning sensation and fever).

PARIṆĀMA ŚŪLA

Nidāna

- स्वैर्निदानैः प्रकुपितो वायुः सन्निहितस्तदा ॥ कफपित्ते समावृत्य शूलकारी भवेद्बली ।
  भुक्ते जीर्यति यच्छूलं तदेव परिणामजम् ॥ (M.N.26/15-16)

The Vāta aggravates due to indulgence of its own causative factors and this aggravated Vāta associated with Kapha and Pitta, later gets obstructed by these doṣas and produces Balī śūla (severe abdominal pain) during Bhukte jīryati (the digestion of food) known as Pariṇāma śūla.

- बलासः प्रच्युतः स्थानात् पित्तेन सह मूर्छितः। वायुमादाय कुरुते शूलं जीर्यति भोजने॥
  कुक्षौ जठरपार्श्वेषु नाभौ बस्तौ स्तनान्तरे। पृष्ठमूलप्रदेशेषु सर्वेष्वेतेषु वा पुनः॥
  भुक्तमात्रेऽथवा वान्ते जीर्णेऽन्ने च प्रशाम्यति। षष्टिकव्रीहिशालीनामोदनेन विवर्धते॥
  तत् परिणामजं शूलं दुर्विज्ञेयं महागदम्। तमाहू रसवहानां स्रोतसां दुष्टिहेतुकम्॥
  केचिदन्नद्रवं प्राहुरन्ये तत् पक्तिदोषतः। पक्तिशूलं वदन्त्येके केचिदन्नविदाहजम् । (Ma.M.N.26/15-16)

Ācārya Vijayarakṣita stated that the Balāsa (Kapha) displaced from its place along with excited Pitta seizing the Vāta produces Śūla jīryati bhojana (severe abdominal pain during the digestion of food) along with pain in the Kukṣau (stomach), Jaṭharapārśveṣu (side of the abdomen), Nābhau (umbilical region), Bastau (bladder region), Stanāntare (in between the breast) and Pṛṣṭhamūla (sacral region).

The pain gets subsided immediately Bhuktemātre (after intake of food) or Vānte (after vomiting) or Jīrṇeanne (after digestion of food) and the pain aggravates due to intake of Ṣaṣṭikavrīhiśālīnāmodanena (Ṣaṣṭika, Vrīhi and Śālī types of rice). This condition is known as Pariṇāma śūla which is caused by Rasavaha srotoduṣṭi and it is considered as Durvijñeya (hardly conceivable) and Mahāgada. Some scholars call it as Annadrava śūla and some call it as Paktiśūla and some others call it as Annavidāhaja śūla due to Paktidoṣataḥ (disturbances in the digestion).

Bheda and Lakṣaṇās

Ācārya Mādhavakara classified Pariṇāma Śūla into five types namely Vātaja, Pittaja, aphaja, Dvidoṣaja and Sannipātaja

taja Pariṇāma Śūla Lakṣaṇās

- आध्मानाटोपविण्मूत्रविबन्धारतिवेपनैः ॥ स्निग्धोष्णोपशमप्रायं वातिकं तद्वदेद्भिषक् । (M.N.26/17-18)

Ādhmāna (distension of abdomen), Āṭopa (gurgling sound in the abdomen), Viṇmūtravibandha (obstruction of faeces and urine), Arati (distress), Vepana (tremor) and Snigdhoṣṇopaśamaprāyaṃ (pain pacified with unctuous and hot therapies) are the clinical features of Vātaja Pariṇāma Śūla.

Pittaja Pariṇāma Śūla Lakṣaṇās

- तृष्णादाहारतिस्वेदं कट्वम्ललवणोत्तरम् ॥ शूलं शीतशमप्रायं पैत्तिकं लक्षयेद्बुधः । (M.N.26/18-19)

Tṛṣṇā (thirst), Dāha (burning sensation), Arati (distress), Sveda (excessive sweating), Kaṭvamlalavaṇottaram (pain aggravates by intake of pungent, sour and saline substances) and Śītaśamaprāyaṃ (pain pacified with cold therapies) are the clinical features of Pittaja Pariṇāma Śūla.

Kaphaja Pariṇāma Śūla Lakṣaṇās

- छर्दिहृल्लाससम्मोहं स्वल्परुग्दीर्घसन्तति ॥ कटुतिक्तोपशान्तं च तच्च ज्ञेयं कफात्मकम् । (M.N.26/19-20)

Chardi (vomiting), Hṛllāsa (nausea), Sammoha (delusion), Svalparug (mild pain), Dīrghasantati (long standing pain) and Kaṭutiktopaśāntaṃ (pain pacified with pungent and bitter substances) are the clinical features of Kaphaja Pariṇāma Śūla.

Dvidoṣaja and Sannipātaja Pariṇāma Śūla Lakṣaṇās

- संसृष्टलक्षणं बुद्ध्वा द्विदोषं परिकल्पयेत् ॥ त्रिदोषजमसाध्यं तु क्षीणमांसबलानलम् । (M.N.26/20-21)

The clinical features of two doṣas or three doṣas appear together in case of Dvidoṣaja and Sannipātaja Pariṇāma Śūla respectively. And if Sannipātaja Pariṇāma Śūla is associated with Kṣīṇa māṃsabalānalam (diminution of muscles, strength and digestive power), then it is considered as Asādhya (incurable).

ANNADRAVA ŚŪLA

- जीर्णे जीर्यत्यजीर्णे वा यच्छूलमुपजायते ॥ पथ्यापथ्यप्रयोगेण भोजनभोजनन च ।
  न शमं याति नियमात् सोऽन्नद्रव उदाहृतः ॥ (अन्नद्रवाख्यशूलेषु न तावत् स्वास्थ्यमश्नुते ।
  वान्तमात्रे जरत्पित्तं शूलमाशु व्यपोहति) ॥ (M.N.26/21-22)

In Annadrava śūla, the continuous pain appears during Jīrṇe Jīryatyajīrṇe vā (digestion or after digestion of food or before digestion) and is not subsided with Pathyāpathyaprayogeṇa (wholesome and unwholesome substances) and Bhojanābhojanena (with food or without food). It is said to be Na tāvat svāsthyamaśnute (incurable) and the pain gets relieved only after Vāntamātrejaratpittaṃ (vomiting of accumulated Pitta).

Difference between Pariṇāma and Annadrava śūla

Factors	Pariṇāma śūla	Annadrava śūla
Site of pain	Kukṣi (stomach), Jaṭharapārśveṣu (side of the abdomen), Nābhi (umbilical region), Basti (bladder region), Stanāntare (in between the breast)	Udara (abdomen), Upajaṭhara (epigastric region)
Time of pain	Jīryati bhojana (during the digestion of food)	the continuous pain appears during Jīrṇe Jīryatyajīrṇe vā (digestion or after digestion of food or before digestion)
Types	Five types namely Vātaja, Pittaja, Kaphaja, Dvidoṣaja and Sannipātaja	Eka Prakāra (one type)
Clinical features	As per the doṣas involved	Continuous pain
Aggravating factors	Intake of Ṣaṣṭika, Vrīhi and Śālī types of rice.	Intake of Pathyāpathyaprayogeṇa (wholesome and unwholesome substances) and Bhojanābhojanena (with food or without food) or any time
Relieving factors	Bhuktemātre (after intake of food) or Vānte (after vomiting) or Jīrṇeanne (after digestion of food)	Vāntamātre jaratpittaṃ (vomiting of accumulated Pitta)
Prognosis	Sannipātaja Pariṇāma Śūla is associated with Kṣīṇa māṃsabalānalam (diminution of muscles, strength and digestive power) is Asādhya (incurable)	Asādhya (incurable)
Modern parlance	Duodenal ulcer	Gastric ulcer

Sthānika Bheda, Saṃprāpti, and Lakṣaṇās of Śūla

Pārśvaśūla

- ⦿ रुणद्धि मारुतं श्लेष्मा कुक्षिपार्श्वव्यवस्थितः ॥ स संरुद्धः करोत्याशु साध्मानं गुड्गुडायनम् ।
  सूचीभिरिव निस्तोदं कृच्छ्रोच्छ्वासी तदा नरः ॥ नान्नं वाञ्छति नो निद्रामुपैत्यर्तिनिपीडितः ।
  पार्श्वशूलः स विज्ञेयः कफानिलसमुद्भवः ॥ (Su.U.42/117-119)

The Kapha located in the Kukṣi (stomach) and Pārśva (flanks) obstructs the passage of Vāta and produces Ādmāna (distension of abdomen), Guḍguḍāyana (gurgling sounds), Sūcībhiriva nistoda (pain as if being pricked by needles), Kṛcchrocchvāsa (difficulty in breathing), Nānnaṃ vāṅchati (no desire for food) and Nā nidrā (not able to sleep) as he is suffering from the above affliction. Such a pain is called Pārśvaśūla which arises due to Vāta and Kapha doṣa.

Kukṣiśūla

- ⦿ प्रकुप्यति यदा कुक्षौ वह्निमाक्रम्य मारुतः ॥ तदाऽस्य भोजनं भुक्तं सोपस्तम्भं न पच्यते ।
  उच्छ्वसित्यामशकृता शूलेनाहन्यते मुहुः ॥ नैवासने न शयने तिष्ठन् वा लभते सुखम् ।
  कुक्षिशूल इति ख्यातो वातादामसमुद्भवः ॥ (Su.U.42/123-125)

The aggravated Vāta afflicts the digestive fire and Bhukta bhojana (the ingested food), gets incarcerated in the region of the Kukṣi (stomach) resulting in Upastambha (Stabdhatānvitam - stiffened) and Na pācyate (indigestion of ingested food), Ucchvasitya (difficulty in breathing), Āmaśakṛtā (passes faeces with undigested food), Muhuḥ śūla (frequent attacks of pain) and Naivāsane na śayane vā labhate sukham (is not relieved either sitting or lying position). This condition is known as Kukṣiśūla which is originated due to Vāta and Āma.

Hṛdśūla

- ⦿ कफपित्तावरुद्धस्तु मारुतो रसमूर्च्छितः ॥ हृदिस्थः कुरुते शूलमुच्छ्वासारोधकं परम् । स हृच्छूल इति ख्यातो रसमारुतसम्भवः ॥
  (Su.U.42/131-132)

Vāta associated with the aggravated Rasa dhātu being obstructed by Kapha and Pitta, gets localized in the heart and produces Hrcchūla (pain in the heart) which is characterized by Śūla (pain) and Ucchvāsārodhakaṃ (difficulty in breathing). Thus the condition is predominantly produced by Rasa dhātu and Vāta doṣa.

Bastiśūla

- ⦿ संरोधात् कुपितो वायुर्बस्तिमावृत्य तिष्ठति ॥ बस्तिवङ्क्षणनाभीषु ततः शूलोऽस्य जायते ।
  विण्मूत्रवातसंरोधी बस्तिशूलः स मारुतात् ॥ (Su.U.42/133-134)

Due to Saṃrodhāt (suppression of natural urges like urine, faeces etc.,), the Vāta gets aggravated and incarcerated in the region of the Basti (urinary bladder) and produces pain in the Bastivaṅkṣaṇanābhiṣu (bladder, groin and umbilical regions). This condition is known as Bastiśūla, which is originated due to Vāta and produces Viṇmūtravātasaṃrodhī (retention of faeces, urine and flatus).

Mūtraśūla

- ⦿ नाभ्यां वङ्क्षणपार्श्वेषु कुक्षौ मेढ्रान्त्रमर्दकः । मूत्रमावृत्य गृह्णाति मूत्रशूलः स मारुतात् । (Su.U.42/135)

The aggravated Vāta produces Mardaka (pounding type of pain) in Nābhyāṃvaṅkṣaṇapārśveṣu kukṣau meḍhrāntra (umbilical, groins, flanks, stomach, penis and intestines) and Mūtramāvṛtya (retention of urine). This condition is known as Mūtraśūla.

Purīṣaśūla

- ⦿ वायुः प्रकुपितो यस्य रूक्षाहारस्य देहिनः । मलं रुणद्धि कोष्ठस्थं मन्दीकृत्य तु पावकम् ॥
  शूलं सञ्जनयंस्तीव्रं स्रोतांस्यावृत्य तस्य हि । दक्षिणं यदि वा वामं कुक्षिमादाय जायते ॥

सर्वत्र वर्धते क्षिप्रं भ्रमन्नथ सघोषवान् । पिपासा वर्धते तीव्रा भ्रमो मूर्च्छा च जायते ॥
उच्चारितो मूत्रितश्च न शान्तिमधिगच्छति । विट्शूलमेतज्जानीयाद्भिषक् परमदारुणम् ॥ (Su.U.42/136-139)

Due to intake of Rūkṣāhārasya dehinaḥ (non-unctuous or dry food substances by the person regularly), the Vāta gets aggravated inturn afflicting the digestive fire and obstructs the Mala (faeces) in the Koṣṭha (abdomen) and produces Tīvraśūla (severe pain) by obstructing the Srotas (channels) in the Dakṣīṇa (right) or Vāma (left) side of the Kukṣi (stomach), but it extends quickly to whole abdomen with rumbling sound associated with Pipāsā vardhate tīvra (excessively increased thirst), Bhrama (giddiness) and Mūrcchā (fainting). Even after passing the Mūtra (urine) and Purīṣa (faeces), there will be no relief. This absolute dreadful condition is known as Viṭśūla or Purīṣaśūla by the physicians.

Avipākādbhavecchūla (Annaja Śūla)

⊙ अतिमात्रं यदा भुक्तं पावके मृदुतां गते । स्थिरीभूतं तु तत्कोष्ठे वायुरावृत्य तिष्ठति ॥
अविपाकगतं ह्यन्नं शूलं तीव्रं करोत्यति । मूर्च्छाऽऽध्मानं विदाहश्च हृदुत्क्लेशो विलम्बिका ॥
विरिच्यते छर्दयति कम्पतेऽथ विमुह्यति । अविपाकाद्भवेच्छूलस्त्वन्नदोषसमुद्भवः ॥ (Su.U.42/142-144)

Due to indulging in Atimātra bhuktaṃ (excessive quantity of food) during Mṛdupāvake (diminished digestive fire), the Vāta gets aggravated and does Sthirībhūta (stagnate the food) and Āvṛtya (obstructs the food) in the Koṣṭha (abdomen). Thus the Avipākagata hyanna (undigested food) produces Tīvraśūla (severe pain) along with Mūrchā (fainting), Ādhmāna (distension of the abdomen), Vidāha (burning sensation), Hṛtkleṣa (nausea) and Vilambikā. And the person has Virecyate (diarrhoea), Chardayati (vomiting), Kampate (tremors) and Vimuhyati (loss of consciousness). This condition is known as Avipākādbhavecchūla which is originated due to Anna doṣa.

Śūla Upadrava

⊙ वेदनातितृषा मूर्च्छा ह्यानाहो गौरवारुची। भ्रमो ज्वरः कृशत्वं च बलहानिस्तथैव च।
कासः श्वासश्च हिक्का च शूलस्थोपद्रवाः स्मृताः॥ (Y.R.29/20)

Vedana (pain), Atitṛṣā (excessive thirst), Mūrcchā (fainting), Ānāha (distension of abdomen and constipation), Gaurava (heaviness), Aruci (anorexia), Bhrama (giddiness), Jvara (fever), Kṛśa (emaciation), Balahāni (loss of strength), Kāsa (cough), Śvāsa (dyspnoea) and Hikkā (hiccup) are the complications of Śūla.

Sādhyāsādhyata of Śūla

⊙ एकदोषोत्थितः साध्यः कृच्छ्रसाध्यो द्विदोषजः। सर्वदोषोत्थितो घोरस्त्वसाध्यो भूर्युपद्रवः॥ (Y.R.29/19)

The Śūla caused by Ekadoṣaja (single doṣa) is Sādhya (curable); caused by Dvidoṣaja (two oṣas) are Kṛcchrasādhya (difficult for cure) and caused by Sarvadoṣaja (all the three doṣas nultaneously) which is Ghora (severe) and associated with Upadrava (complications) is ādhya (incurable).

In Contemporary science,

Śūla roga can be predominantly characterised by conditions like peptic ulcer in contemporary science. Peptic ulcer is characterized by discontinuation in the inner lining of the gastrointestinal (GI) tract and it extends into muscularis propria layer of the gastric epithelium as a result of gastric acid and pepsin secretion. It usually occurs in the stomach and proximal duodenum and it may involve the lower esophagus and Meckel's diverticulum. An ulcer developed in the stomach is known as gastric ulcer whereas in the first part of the intestines is known as a duodenal ulcer.

Etiology: Helicobacter pylori infection and NSAIDs are the main causative factors. Other causes include hereditary factors, dietary habits, mental stress, heavy smoking, excessive alcohol consumption and medications like corticosteroids, aspirin and anti-rheumatic drugs.

Clinical features: Recurrent abdominal pain is the main symptom of peptic ulcer disease (both in gastric and duodenal ulcer). Other symptoms include bloating and distension of abdomen, heartburn and nausea or vomiting. And in severe cases, increased severity of the pain, pallor, blood vomiting, dark or black stools (malena) are seen.

The differentiating features between the duodenal and gastric ulcer are, the burning or gnawing epigastric pain or hunger pain occurs 90 min to 3 hours after a meal and is frequently relieved after intake of food or antacids resulting in weight gain. And the pain awakes the patient from sleep around 2 AM in the night in case of duodenal ulcer. Whereas in gastric ulcer, the burning or gnawing epigastric pain occurs after meals but in many cases it is indistinguishable from that of duodenal ulcer. Anorexia, nausea or vomiting and significant weight loss may be present.

Complications: Upper gastrointestinal bleeding, perforation, pancreatitis (due to posterior penetration), gastric outlet obstruction or pyloric obstruction and malignancy (especially with gastric ulcer)

Diagnosis: Diagnosis is based on the clinical history, endoscopy, imaging studies, serum gastrin levels and helicobacter testing. Clinically, the history should be taken properly and noted for the presence of any complications. Endoscopy is the gold standard for diagnosing the peptic ulcer and imaging studies like Barium meal x-ray is less sensitive but it is indicated when endoscopy is contraindicated and CT scan helps in diagnosing the perforation and gastric outlet obstruction. Fasting serum gastrin levels are elevated in Zollinger-Ellison syndrome. Testing the Helicobacter pylori infection is more essential in peptic ulcers and it can be made by urea breath test, stool antigen test, rapid urease test and urine based ELISA and also antibodies to H.pylori can be measured.

MCQ's

1. Śūla produces discomfort like that of

a) Nirghāta Vedana b) Kīlaka nikhānita

c) Ropaṇavatpīḍa d) All of the above

2. According to Mādhavakara, Śūla is of .... types

a) 5 b) 6

c) 7 d) 8

3. According to Suśruta, Śūla is of .... types

a) 4 b) 5

c) 6 d) 8

4. According to Suśruta, Śūla is of .... types

a) 4 b) 5

c) 6 d) 8

5. मध्यन्दिने कुप्यति चार्धरात्रे निदाघकाले is seen in

a) Vātaja Śūla b) Pittaja Śūla

c) Kaphaja Śūla d) Sannipātaja Śūla

6. भुक्ते सदैव हि रुजं कुरुतेऽतिमात्रं is seen in

a) Vātaja Śūla b) Pittaja Śūla

c) Kaphaja Śūla d) Sannipātaja Śūla

7. दाहज्वरकरो घोरो is seen in

a) Kaphavātaja Śūla b) Kaphapittaja Śūla

c) Vātapittaja Śūla d) Sannipātaja Śūla

8. भुक्ते जीर्यति यच्छूलं is the feature of

a) Annadrava Śūla b) Āmaja Śūla

c) Kaphaja Śūla d) Pariṇāma Śūla

Pariṇāma Śūla is of .... types

a) 3 b) 4

c) 5 d) 6

10. In Pariṇāma Śūla, the pain gets subsided immediately after

a) Bhuktemātre b) Vānte

c) Jīrṇeanne d) All of the above

11. जीर्णे जीर्यत्यजीर्णे वा यच्छूलं denotes

a) Annadrava Śūla b) Āmaja Śūla

c) Kaphaja Śūla d) Pariṇāma Śūla

12. Which type of Śūla gets aggravated after intake of Ṣaṣṭika, Vrīhi and Śālī types of rice

a) Annadrava Śūla b) Āmaja Śūla

c) Kaphaja Śūla d) Pariṇāma Śūla

13. Doṣa involved in Pārśvaśūla

a) Vātakapha b) Vātapitta

c) Kaphapitta d) Sannipātaja

14. नैवासने न शयने तिष्ठन् वा लभते सुखम् is the feature of

a) Pariṇāma Śūla b) Hṛdśūla

c) Kukṣiśūla d) Pārśvaśūla

15. Which infection is a main causative factor of peptic ulcers?

a) Epstein-Barr Virus b) Helicobacter pylori

c) Varicella-Zoster Virus d) Dengue virus

16. Hunger pain is seen in

a) Gastric ulcer b) Duodenal ulcer

c) Pancreatitis d) Both a and b

17. Which infection is a main causative factor of peptic ulcers?

a) Epstein-Barr Virus b) Helicobacter pylori

c) Varicella-Zoster Virus d) Dengue virus

Answer Keys

1	d	2	d	3	a	4	a	5	b	6	c	7	c	8	d	9	c
10	d	11	a	12	d	13	a	14	c	15	a	16	b	17	a		

Short Answer Questions

1. Explain Śūla Nidāna and Saṃprāpti
2. Explain Pariṇāma Śūla
3. Explain Annadrava Śūla

Long Answer Questions

1. Explain Nidāna, Saṃprāpti, bheda and Lakṣaṇās of Śūla
2. Define and differentiate the Pariṇāma Śūla and Annadrava Śūla

◆

TOPIC 5

Atisāra and Pravāhika

ATISĀRA

Introduction

Atisāra is one of the diseases of Purīṣavaha srotas. The disease Atisāra has been named on the basis of its Pratyātma Lakṣaṇa (cardinal symptom) i.e. Atisaraṇa of Mala (excessive excretion of Mala). Ācārya Vāgbhaṭa explained that Grahaṇī doṣa and Atisāra are similar in their causative factors. Even Ācārya Caraka described the same i.e. Agnibalahāni (diminution of digestive power) is the causative factor for both Grahaṇī doṣa and Atisāra. But Ācārya Suśruta described Atisāra after Jvara because Agni upaghāta pūrvaka (destruction of digestive fire) appears first in both the diseases.

Nirūkti and Paribhāṣā

- अत्यन्तं सारयति मलम् इति । (Vācaspatyam)

Excessive expulsion of faecal matter is called Atisāra

- अतिशयेन मलं द्रवीकृत्य सरति निःसारयति अति । (SKD)
- अतिसारयति मलम् द्रवीकृत्य अति । रोगभेदे उदारामये । (Vācaspatyam)

Excessive expulsion of liquid faecal matter is called Atisāra. Udārāmaya is the synonym of Atisāra.

- सरणलक्षणोऽतीसारः। (Ḍa.Su.Sū.21/34)

That which does Saraṇa (oozing) is called as Atisāra.

- गुदेन बहुद्रवसरणमतिसार इत्यर्थः। (Ma.M.N.3/4)
- अतीव सरतीत्यतीसारो गुदेन । (Āḍ.Śā.P.7/7-8)

Excessive expulsion of liquid faecal matter through Guda (anal region) is called Atisāra.

- अतिसरणम् अतीसारः, । अतिरत्यर्थवचने, सरतिर्गतिकर्मणि। तस्मात्तदत्यन्तसरणादतीसार इति स्मृतः" इति। (Ḍa.Su.U.40/1-2)

Atisaraṇa (excessive oozing out) is called Atisāra. The word 'Ati' means Atyartha refers to excessive or extreme and 'Saraṇa' means Gatikarma refers to action of movement. Hence Atyantasaraṇāt (excessive flow of liquid matter from the anus) is called Atisāra.

Sāmānya Nidāna and Samprāpti of Atisāra

- गुर्वतिस्निग्धरूक्षोष्णद्रवस्थूलातिशीतलैः । विरुद्धाध्यशनाजीर्णैरसात्म्यैश्चापि भोजनैः ॥
  स्नेहाद्यैरतियुक्तैश्च मिथ्यायुक्तैर्विषाद्भयात् । शोकाद्दुष्टाम्बुमद्यातिपानात् सात्म्यर्तुपर्ययात् ॥
  जलातिरमणैर्वेगविघातैः कृमिदोषतः । नृणां भवत्यतीसारो लक्षणं तस्य वक्ष्यते । (Su.U.40/3-5)

Excessive indulgence in Guru (heavy substances), Snigdha (unctuous substances), Rūkṣa (dry substances), Uṣṇa (hot substances), Drava (liquid substances), Sthūla (solid substances), Atiśītalai (excessive cold substances) and Viruddha (incompatible substances); Adhyaśana (eating in excess), Ajīrṇa (indigestion) and Asātmya bhojana (unaccustomed substances); Atimithyayukta Snehādikarma (excessive or improper use of oleation, sudation etc., therapies), Viṣa (poison), Bhayāt (fear), Śoka (grief), Duṣṭāmbu pānāt (drinking impure water), Madyātipānāt (excessive drinking of alcohol), Sātmyarṭuparyayāt (indulging against the seasonal regimens), Jalātiramaṇai (excessive playing in water), Vegavighātai (suppression of natural urges) and Kṛmi doṣa (worm infestation) are the causative factors of the disease Atisāra.

- संशम्यापान्धातुरन्तः कृशानुं वर्चोमिश्रो मारुतेन प्रणुन्नः । वृद्धोऽतीवाधः सरत्येष यस्माद्व्याधिं घोरं तं त्वतीसारमाहुः ॥ (Su.U.40/6)

Due to the above said causative factors, the aggravated Vāta dislodges the Āp dhātu (liquid aspects of the body) to the Koṣṭha, and this Āp dhātu does the Saṃśamyāntaḥ Kṛśānu (diminishes the internal digestive fire), Varcomiśro (mixes with the faecal matter) and liquefies and increases its quantity and expels them excessively through the anus. This Ghora vyādhi (serious disease) is known as Atisāra.

Samprāpti Ghaṭaka of Atisāra:

Doṣa	Vāta Pradhāna Tridoṣa
Dūṣya	Āpāṃ dhātus (Kāyadravaḥ)
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Agni māndhya janya Āma
Srotas	Purīṣa, Anna, Sveda and Udaka vaha
Srotoduṣṭi	Atipravṛtti,
Sañcāra sthāna	Koṣṭha
Udbhava sthāna	Āmapakvāśaya
Vyakta sthāna	Guda
Adhiṣṭhāna	Mahāsrotas
Rogamārga	Abhyantara

Pūrvarūpa of Atisāra

- तोदो हृद्गुदकोष्ठेषु गात्रसादो मलग्रहः। आध्मानमविपाकश्च .... । (A.H.Ni.8/5)

Hṛdgudakoṣṭha toda (pricking pain in the region of heart, rectum and alimentary tract), Gātra sāda (weakness of the body), Mala graham (constipation), Ādhmāna (distension of abdomen) and Avipāka (indigestion) are the premonitory clinical features of Atisāra.

- हृन्नाभिपायूदरकुक्षितोदगात्रावसादानिलसन्निरोधाः ॥ विट्सङ्ग आध्मानमथाविपाको भविष्यतस्तस्य पुरःसराणि । (Su.U.40/8-9)

Hṛnnābhipāyūdarakukṣitoda (piercing pain in the regions of the heart, umbilicus, rectum, abdomen and sides of the abdomen), Gātrāvasāda (fatigue of the body), Anilasannirodhaḥ (obstruction to the Vāta), Viṭsaṅga (constipation), Ādhmāna (distension of abdomen) and Avipāka (indigestion) are the premonitory symptoms of the disease Atisāra.

Atisāra Bheda

- एकैकशः सर्वशश्चापि दोषैः शोकेनान्यः षष्ठ आमेन चोक्तः । (Su.U.40/7)
- दोषैर्व्यस्तैः समस्तैश्च भयाच्छोकाच्च षड्विधः। अतीसारः ... ॥ (A.H.Ni.8/1)

Ācārya Suśruta classified Atisāra into six types namely Vātaja, Pittaja, Kaphaja, Sannipātaja, Śokaja and Āmaja. Ācārya Vāgbhaṭa and Caraka described Bhayaja Atisāra instead of Āmaja Atisāra.

- पृथक्त्रिदोषैः सर्वैश्च शोकादामाद्भयादपि। अतीसारः सप्तधा॥ (Śā.P.7/7)

Ācārya Śārṅgadhara classified Atisāra into seven types namely Vātaja, Pittaja, Kaphaja, Sannipātaja, Śokaja, Āmaja and Bhayaja.

Atisāra	Caraka	Suśruta	Vāgbhaṭa	Śārṅgadhara
Vātaja	+	+	+	+
Pittaja	+	+	+	+
Kaphaja	+	+	+	+
Sannipātaja	+	+	+	+
Śokaja	+	+	+	+
Bhayaja	+	-	+	+
Āmaja	-	+	-	+

- ⊙ शकृद्वातमसृक् पित्तं कफं वा योऽतिसार्यते। पक्वं , तत्र स्ववर्गीयैर्बस्तिः श्रेष्ठं भिषग्जितम्॥
षण्णामेषां द्विसंसर्गात् त्रिंशद्भेदा भवन्ति तु। केवलैः सह षट्त्रिंशद्विद्यात् सोपद्रवानपि॥ (Ca.Si.8/20-21)

Ācārya Caraka in Siddhi sthāna described that the six types (Āmaja, Purīṣaja, Vātaja, Raktaja, Pittaja and Kaphaja) of Atisāra may get manifested in the form of a combination of two types, thus making thirty more varieties. By adding the main six types, thirty-six varieties of Atisāra are manifested along with their complications.

- ⊙ अतीसारः समासेन द्विधा सामो निरामकः। सासृङ्निरसः .. । (A.H.Ni.8/13)

Ācārya Vāgbhaṭa classified Atisāra into two types namely Āmātisāra (Sāmātisāra) and Nirāmātisāra. Again, he classified Atisāra into two types: Sāsra (mixed with blood) and Nirasra (not mixed with blood).

Viśeṣa Nidāna, Saṃprāpti and Lakṣaṇās of Atisāra

Vātaja Atisāra

- ⊙ अथावरकालं वातलस्य वातातपव्यायामातिमात्रनिषेविणो रूक्षाल्पप्रमिताशिनस्तीक्ष्णमद्य व्यवायनित्यस्योदावर्तयतश्च वेगान् वायुः प्रकोपमापद्यते, पक्ता चोपहन्यते; स वायुः कुपितोऽग्नावुपहते मूत्रस्वेदौ पुरीषाशयमुपहृत्य, ताभ्यां पुरीषं द्रवीकृत्य, अतीसाराय प्रकल्पते। तस्य रूपाणि- विज्जलमामं विप्लुतमवसादि रूक्षं द्रवं सशूलमामगन्धमीषच्छब्दमशब्दं वा विबद्धमूत्रवातमतिसार्यते पुरीषं, वायुश्चान्तःकोष्ठे सशब्दशूलस्तिर्यक् चरति विबद्ध इत्यामातिसारो वातात्। पक्वं वा विबद्धमल्पाल्पं सशब्दं सशूलफेनपिच्छापरिकर्तिकं हृष्टरोमा विनिःश्वसञ् शुष्कमुखः कट्यूरुत्रिकजानुपृष्ठपार्श्वशूली भ्रष्टगुदो मुहुर्मुहुर्विग्रथितमुपवेश्यते पुरीषं वातात्; तमाहुरनुग्रथितमित्येके, वातानुग्रथितवर्चस्त्वात्॥ (Ca.Ci.19/5)

When Vātaja Prakṛti person indulges in Vātāpavyāyāmātimātra niṣevaṇa (excessive exposure to wind, sun heat and physical exercise), Rūkṣālpapramitāśina (consumes non-unctuous diets and less or limited quantities of diet), Tīkṣṇamadya (sharp or strong alcoholic drinks), Vyavāyanityasya (indulges daily in sexual intercourse) and Vegānudāvartayata (suppression or reversion of manifested urges). By these factors, Vāta aggravates and afflicts the Agni (digestive power). This aggravated Vāta after reducing the Agni (digestive power), forcefully pulls the Mūtra (urine) and Sveda (sweat) to the Purīṣāśaya (colon) and liquefies the faeces and produces Vātaja Atisāra.

The clinical features of Vātaja Atisāra are – in association with Āma, the patient passes Vijjala (slimy), Vipluta (scattered in the water), Avasādi (when the faeces placed over the earth, it disappear due to being soaked), Rūkṣaṃ dravaṃ (dry and watery faeces) Saśūla (passed with pain), Āmāmagandhamīṣacchabdamaśabdaṃ (has stinking smell and passes with less or no sound), Vibaddhamūtravātamatisāryate (passing faeces is not accompanied by urine and flatus) and this obstructed Vāyu (flatus) moves in the abdomen obliquely along with Sasabdaśūla (sound and pain).

Vibaddhamalpālpaṃ (constipated or passes small quantity repeatedly) with Saśabdaṃ saśūlaphenapicchāparikartika (sound, colicky pain, frothy, slimy and cutting pain), Hṛṣṭaromā (horripilation), Viniḥśvasañ (producing sound while respiring), Śuṣkamukhaḥ (dryness of the mouth) Kaṭhyūrutrikajānupṛṣṭhapārśvaśūlī (pain in the waist, thighs, sacral, knees, back and flanks) Bhraṣṭaguda (prolapse of the rectum) and Muhurmuhurvigrathitamupaveśyat purīṣaṃ vātāt (passes repeated scybalous or nodular faeces due to Vāta) are the clinical features of Pakva Vātaja Atisāra. Some scholars call this as Anugrathita Atisāra due to expulsion of scybalous faeces.

- ⊙ शूलाविष्टः सक्तमूत्रोऽन्त्रकूजी स्रस्तापानः सन्नकट्यूरुजङ्घः ॥ वर्चो मुञ्चत्यल्पमल्पं सफेनं रूक्षं श्यावं सानिलं मारुतेन । (Su.U.40/9-10)

- ⊙ अरुणं फेनिलं रूक्षमल्पमल्पं मुहुर्मुहुः । शकृदामं सरुक्शब्दं मारुतेनातिसार्यते ॥ (M.N.3/6)

- ⊙ तत्र वातेन विड्जलम्॥ अल्पाल्पं शब्दशूलाढ्यं विबद्धमुपवेश्यते। रूक्षं सफेनमच्छं च ग्रथितं वा मुहुर्मुहुः॥ तथा दग्धगुडाभासं सपिच्छापरिकर्तिकम्। शुष्कास्यो भ्रष्टपायुश्च हृष्टरोमा विनिष्टनन्। (A.H.Ni.8/5-7)

Śūlāviṣṭaḥ (colic pain), Saktamūtra (suppression of urine), Antrakūjī (gurgling sound in the intestines), Srastāpānaḥ (looseness of the Apāna vāta), Sannakaṭyūrujaṅgaḥ (feeling of tiredness in the waist, thighs and calf regions) and Varcomuñcatyalpalpaṃ saphenaṃ rūkṣaṃ śyāvaṃ sānilaṃ (passes scanty faeces frequently along with frothy and is dry, dark brown coloured and with flatus) are the clinical features of the Vātaja Atisāra.

Pittaja Atisāra

- ⊙ पित्तलस्य पुनरम्ललवणकटुकक्षारोष्णतीक्ष्णातिमात्रनिषेविणः प्रतताग्निसूर्यसन्तापोष्ण मारुतोपहतगात्रस्य क्रोधेर्ष्याबहुलस्य पित्तं प्रकोपमापद्यते। तत् प्रकुपितं द्रवत्वादूष्माणमुपहत्य पुरीषाशयविसृतमौष्ण्याद् द्रवत्वात् सरत्वाच्च भित्त्वा पुरीषमतिसाराय प्रकल्पते। तस्य रूपाणि- हारिद्रं हरितं नीलं कृष्णं रक्तपित्तोपहितमतिदुर्गन्धमतिसार्यते पुरीषं, तृष्णादाह स्वेदमूर्च्छाशूलब्रध्नसन्तापपाकपरीत इति पित्तातिसारः॥ (Ca.Ci.19/6)

When Pittaja Prakṛti person indulges in Amlalavaṇakaṭuka kṣāroṣṇatīkṣṇātimātra niṣeviṇaḥ (excessive intake of sour, saline, pungent, alkaline, hot and sharp substances), Pratatāgnisūryasantāpoṣṇamārutopahata (exposure to fire, burning sun and hot winds for a long time) and Krodherṣyābahulasya (afflicted with excessive anger and envy), the Pitta gets aggravated and it suppresses the Agni by its Dravatva (liquidity) and reaches the Purīṣāśaya (colon), disintegrates the faeces because of its Uṣṇatva (hotness), Dravatvāt (liquidity) and Saratvāt (mobility) qualities, thus produces Pittaja Atisāra.

The clinical features of Pittaja Atisāra are the patient passes faeces as Hāridraṃ (yellow), Haritaṃ (green), Nīlaṃ (blue), Kṛṣṇaṃ (black) in colour associated with Raktapittopahita (blood and bile), which is Atidurgandha (extremely foul smelling). Tṛṣṇā (thirst), Dāha (burning sensation), Sveda (sweating), Mūrcchā (fainting), Śūla (colic pain) and Bradhna santāpapāka (inflammation and suppuration of anus) are the associated clinical features of Pittaja Atisāra.

- दुर्गन्ध्युष्णं वेगवन्मांसतोयप्रख्यं भिन्नं स्विन्नदेहोऽतितीक्ष्णम् ॥ पित्तात् पीतं नीलमालोहितं वा तृष्णामूर्च्छादाहपाकज्वरार्तः। (Su.U.40/10-11)
- पित्तेन पीतमसितं हारिद्रं शाद्वलप्रभम्। सरक्तमतिदुर्गन्धं तृण्मूर्च्छास्वेददाहवान्॥
  सशूलं पायुसन्तापपाकवान् । (A.H.Ni.8/8-9)

The clinical features of Pittaja Atisāra are the patient passes faeces as Durgandha (foul smell), Uṣṇa (hot), Vegavanmāṃsatoyaaprakhyaṃ (loose bowels resembling the washing of meat), Bhinna (split in nature) and Pītaṃ nīlamālohita (yellowish, bluish or reddish in colour); and it is associated with Svinnadeho (sweats excessively all over the body), Aitikṣṇam (feels an excessive sharp sensation in the anal region), Tṛṣṇā (thirst), Mūrcchā (fainting), Dāha (burning sensation), Pāka (suppuration of affected areas) and Jvara (fever).

Kaphaja Atisāra

- श्लेष्मलस्य तु गुरुमधुरशीतस्निग्धोपसेविनः सम्पूरकस्याचिन्तयतो दिवास्वप्नपरस्यालसस्य श्लेष्मा प्रकोपमापद्यते। स स्वभावाद् गुरुमधुरशीतस्निग्धः स्रस्तोऽग्निमुपहत्य सौम्यस्वभावात् पुरीषाशयमुपहत्योपक्लेद्य पुरीषमतिसाराय कल्पते। तस्य रूपाणि- स्निग्धं श्वेतं पिच्छिलं तन्तुमदामं गुरु दुर्गन्धं श्लेष्मोपहितमनुबद्धशूलमल्पाल्पमभीक्ष्णमतिसार्यते सप्रवाहिकं, गुरूदरगुदबस्तिवङ्क्षणदेशः कृतेऽप्यकृतसञ्ज्ञः सलोमहर्षः सोत्क्लेशो निद्रालस्यपरीतः सदनोऽन्नद्वेषी चेति श्लेष्मातिसारः॥ (Ca.Ci.19/7)

When Kaphaja Prakṛti person indulges in Gurumadhuraśītasnigdhopasevinaḥ sampūrakasyā (excessive consuming of heavy, sweet, cold and unctuous substances), Acintayato (free from worries) and Divāsvapnapara (day sleeping regularly) and Alasasya (remains indolent), the Kapha aggravates and moves downwards due to its Guru (heavy), Madhura (sweet), Śīta (cold) and Snigdha (unctuous) qualities and afflicts the Agni by its Saumya svabhāva (natural cooling quality) and reaches the Purīṣāśaya (colon), liquefies the faeces because of its Utkleda (moistening) quality, thus produces Kaphaja Atisāra.

The clinical features of Kaphaja Atisāra are the patient passes Snigdha (unctuous), Śveta (white), Picchila (slimy), Tantumada (fibrous), Āmaṃ (undigested), Guru (heavy), Durgandha (foul smelling), Śleṣmopahita (mixed with mucous), Anubaddha Śūla (continuous colic pain), Alpālpabhīkṣṇamatisāryate sapravāhikaṃ (defecates little quantity of faeces frequently with tenesmus), Gurūdaragudabastivaṅkṣaṇadeśaḥ (heaviness in the abdomen, anal, bladder and groin region), Kṛteapyakṛtasaṃjñaḥ (defecates without knowledge), Salomaharṣaḥ (horripilation), Utkleśa (nausea), Nidra (over sleepiness), Alasyaparītaḥ (indolence), Sadana (fatigue) and Annadveṣī (dislike for food)

- तन्द्रानिद्रागौरवोत्क्लेशसादी वेगाशङ्की सृष्टविट्कोऽपि भूयः ॥
  शुक्लं सान्द्रं श्लेष्मणा श्लेष्मयुक्तं भक्तद्वेषी निःस्वनं हृष्टरोमा । (Su.U.40/11-12)
- ....श्लेष्मणा घनम्। पिच्छिलं तन्तुमच्छ्वेतं स्निग्धमामं कफान्वितम्॥
  अभीक्ष्णं गुरु दुर्गन्धं विबद्धमनुबद्धरुक्। निद्रालुरलसोऽन्नद्विडल्पाल्पं सप्रवाहिकम्॥
  सरोमहर्षः सोत्क्लेशो गुरुबस्तिगुदोदरः। कृतेऽप्यकृतसंज्ञश्च...। (A.H.Ni.8/9-11)

The clinical features of Kaphaja Atisāra are Tandrā (drowsiness), Nidrā (excessive sleep), Gaurava (heaviness of the body), Utkleśa (nausea), Sādī (depression), Vegaśaṅkī sṛṣṭaviṭoapi bhūyaḥ (after defecation patient feels the desire to pass the faeces again), Bhaktadveṣī (dislike for food) and Hṛṣṭaromā (horripilation); and patient passes faeces with Śuklaṃ (whitish in colour), Sāndra (viscid in consistency) and Śleṣma (mixed with mucus) associated with Niḥsvana (Niḥśabda - without any sound or soundless).

Atisāra	Vātaja Atisāra	Pittaja Atisāra	Kaphaja Atisāra
Śārīrika Lakṣaṇa	Śūlāviṣṭaḥ, Saktamūtra, Antrakūjī, Sannakaṭyūrujaṅgaḥ	Tṛṣṇā, Mūrcchā, Dāha, Pāka, Jvara	Tandrā, Nidra, Gaurava, Utkleṣa, Sādī, Bhaktadveṣī, Hṛṣṭaromā
Purīṣaja Lakṣaṇa	Alpālpa, Muhurmuhu, Saśabda	Durgandha, Uṣṇa, Māṃsatoyābhaṃ Svinnadeha	Vegaśaṅkī sṛṣṭaviṭoapi bhūyaḥ, Sāndra, Śleṣma, Niḥsvana
Colour of Purīṣa	Aruṇa, Śyāva,	Pīta, Nīla, Lohita	Śuklaṃ

Sannipātaja Atisāra

- अतिशीतस्निग्धरूक्षोष्णगुरुखरकठिनविषमविरुद्धासात्म्यभोजनादभोजनात्कालातीतभोजनाद्यत्किञ्चिदभ्यवहरणात्प्रदुष्टमद्यपानीयपानादतिमद्यपानादसंशोधनात् प्रतिकर्मणां विषमगमनादनुपचाराज्ज्वलनादित्यपवनसलिलातिसेवनादस्वप्ना- दतिस्वप्नाद्वेगविधारणादृतु विपर्ययादयथाबलमारम्भाद्भयशोकचित्तोद्वेगातियोगात् कृमिशोषज्वरार्शोविकारातिकर्षणाद्वा व्यापन्नाग्नेस्त्रयो दोषाः प्रकुपिता भूय एवाग्निमुपहत्य पक्वाशयमनुप्रविश्यातीसारं सर्वदोषलिङ्गं जनयन्ति॥ (Ca.Ci.19/8)

Due to intake of Atiśītasnigdharūkṣoṣṇāgurukharakaṭhina (excessively cold, unctuous, non-unctuous, hot, heavy, coarse and hard substances); Viṣama (habitual of intake of irregular meals), Viruddha (foods having mutually contradictory properties), Asātmya bhojanāt (unwholesome food), Abhojanāt (avoidance of food intake), Kālātītabhojanāt (intake of food beyond the proper time), Yatkiñcitdabbhyavaharaṇāt (intake of wholesome and unwholesome substances), Praduṣṭamadyapānīyapānāt (drinking of polluted or defective alcoholic drinks and other drinks), Atimadyapānāt (intake of excessive alcohol), Asaṃśodhanāt (not resorting to purification therapies during appropriate seasons), Pratikarmaṇāṃ (improper administration of therapeutic measures), Viṣamagamanādanupacārā (irregular application of therapeutic measures), Jvalanādityapavanasalilātisevana (excessive exposure to fire, sun, wind and water), Asvapnādatisvapnāt (not sleeping or excessive sleep), Vegavidhāraṇa (suppression of natural urges), Ṛtuviparyayād (not resorting to appropriate regimens during seasons), Ayathābalamārambhat (exertion not in accordance with one's strength), Bhayaśokācittodvegāt (excessive fear, grief and psychological excitation), Kṛmiśoṣajvararśovikārātikarṣaṇāt (extreme emaciation due to worm, phthisis, fever or piles etc.,), the Tridoṣas gets aggravated and afflicts the Agni and reaches the Purīṣāśaya (colon) and produces Sannipātaja Atisāra.

◉ अपि च शोणितादीन् धातूनतिप्रकृष्टं दूषयन्तो धातुदोषस्वभावकृतानतीसारवर्णानुपदर्शयन्ति। तत्र शोणितादिषु धातुष्वतिप्रदुष्टेषु हारिद्रहरितनीलमाञ्जिष्ठमांसधावनसन्निकाशं रक्तं कृष्णं श्वेतं वराहभेदःसदृशमनुबद्धवेदनमवेदनं वा समासव्यत्यासादुपवेश्यते शकृद् ग्रथितमामं सकृत्, सकृदपि पक्वमनतिक्षीणमांसशोणितबलो मन्दाग्निर्विहतमुखरसश्च; तादृशमातुरंकृच्छ्रसाध्यंविद्यात्।एभिर्वर्णैरतिसार्यमाणंसोपद्रवमातुरमसाध्योऽयमितिप्रत्याचक्षीत;तद्यथा-पक्वशोणिताभं यकृत्खण्डोपमं मेदोमांसोदकसन्निकाशं दधिघृतमज्जतैलवसाक्षीर वेसवाराभमतिनीलमति रक्तमतिकृष्णमुदकमिवाच्छं पुनर्मेचकाभमतिस्निग्धं हरितनीलकषायवर्णं कर्बुरमाविलं पिच्छिलं तन्तुमदामं चन्द्रकोपगतमतिकुणपपूतिपूयगन्ध्या माममत्स्यगन्धि मक्षिकाकान्तं कुथितबहुधातुस्रावमल्पपुरीषमपुरीषं वाऽतिसार्यमाणं तृष्णादाहज्वरभ्रमतमकहिक्का श्वासानुबन्धमतिवेदनमवेदनं वा स्रस्तपक्वगुदं पतितगुदवलिं मुक्तनालमतिक्षीणबलमांसशोणितं सर्वपर्वास्थिशूलिनमरोचकारतिप्रलापसम्मोहपरीतं सहसोपरतविकारमतिसारिणमचिकित्स्यं विद्यात्; इति सन्निपातातिसारः॥ (Ca.Ci.19/9)

Due to the causative factors, the aggravated Tridoṣas causes excessive vitiation of the Raktādi dhātus, then the colour of faeces become as per characters of aggravated doṣas and vitiated dhātus. If the Raktādi dhātus are excessively vitiated, then the clinical features are the patient passes faeces as Hāridra (yellow), Harita (green), Nīla (blue), Mañjiṣṭhā (colour similar to Mañjiṣṭhā), Māṃsadhāvana sannikāśaṃ (colour resembles the washing of meat), Raktaṃ (red), Kṛṣṇaṃ (black), Śveta (white) and Varāhamedaḥsadṛśam (resembling the colour of pig-fat) and Anubaddhavedanamavedanaṃ (with or without pain), all these features manifest either combined together or separately.

At times, the patient may pass Grathita (scabalous faeces) and associated with Āma (undigested food) and sometimes associated with Pakva (digested food). Anatikṣīṇamāṃsaśoṇitabala (does not have over depletion of muscle tissue, blood and strength) and Mandāgnirvihatamukharasaśca (suppressed digestive fire with affliction of taste in the mouth). Such patients should be considered as Kṛcchrasādhya (difficult to cure).

The patient becomes Asādhya (incurable) if he passes faeces associated with ollowing characters and complications. The characters of the faeces are resembling the akvaśoṇitābhaṃ (cooked blood), Yakṛtkhaṇḍopamaṃ (liver pieces), Medamāṃsodaka annibhaṃ (resembles the fat and meat washings), Dadhighṛtamajjatailavasākṣīra (resembles e curd, ghee, marrow, oil, fat and milk), Vesavārābham (resembles boneless minced meat) inīlamatiraktamatikṛṣṇam (excessively blue, red or black), Udakamivācchaṃ (clear like ter), Mecakābham (dark blue colour), Atisnigdha (over unctuous), Haritanīlakaṣāyavarṇa een, blue or brown colour), Karbura (variegated colours), Avilaṃ (turbid), Picchilaṃ ny), Tantum (fibrous), Āmaṃ (mixed with undigested food), Candrakopagta (resembles

the light around the moon), Atikuṇapapūtipūyagandhyāma (has excessive bad smell like that of dead body or putrefied smell or smell of pus or smell of undigested food), Matsyagandhi (smell of uncooked fish), Makṣikākāntaṃ (attracted by flies), Kuthitabahudhātusrāvam (has profuse discharge of necrosed dhātus), Alpapurīṣamapurīṣaṃvāatisāryamāṇaṃ (has little or no faecal matter in Atisāra) and he suffers from the complications like Tṛṣṇā (morbid thirst), Dāha (burning sensation), Jvara (fever), Bhrama (giddiness), Tamaka (feeling of darkness before eyes), Hikkā (hiccup), Śvāsa (dyspnoea) and Anubandhamativedanamavedanaṃ (associated with pain or without pain), Srastapakvagudaṃ (prolapse and inflammation of anal canal), Patitaagudavaliṃ muktanālam (displacement or loss of control of anal sphincters); Atikṣīṇabalamāṃsaśoṇitaṃ (excessive loss of strength, muscle tissue and blood), Sarvaparvāsthiśūlinam (pain in all the joints and bones), Arocaka (anorexia), Atipralāpasaṃmohaparītaṃ (excessive delirium and delusion) and Sahasoparatavikāra (sudden disappearance of all the features). Such patients should be considered as Acikitsyaṃ (untreatable), thus is Sannipātaja Atisāra.

◉ तन्द्रायुक्तो मोहसादास्यशोषी वर्चः कुर्यान्नैकवर्णं तृषार्तः ॥ सर्वोद्भूते सर्वलिङ्गोपपत्तिः कृच्छ्रश्चायं बालवृद्धेष्वसाध्यः । (Su.U.40/12-13)

◉ ... सर्वात्मा सर्वलक्षणः॥ (A.H.Ni.8/11)

The clinical features of Tridoṣaja Atisāra are Tandra (drowsiness), Moha (fainting), Sādā (lassitude), Āsyaśoṣī (dryness of the mouth), Tṛṣārta (morbid thirst) and he passes Varcaḥ kuryānnaikavarṇaṃ (faeces of various colours) appears with all the features. It is Kṛcchrasādhya (difficult to cure) and becomes Asādhya (incurable) if occurred in Bāla (child) and Vṛddha (aged persons).

Āgantuja Atisāra

◉ आगन्तू द्रावतीसारौ मानसौ भयशोकजौ। तत्तयोर्लक्षणं वायोर्यदतीसारलक्षणम्॥ (Ca.Ci.19/11)

Āgantuja Atisāra (exogenous diarrhoea) is of psychic origin and is of two types namely Bhayaja (fear) and Śokaja (grief). Their clinical features are similar to those of the Vātaja Atisāra.

◉ भयेन क्षोभिते चित्ते सपित्तो द्रावयेच्छकृत्। वायुस्ततोऽतिसार्येत क्षिप्रमुष्णं द्रवं प्लवम्॥
वातपित्तसमं लिङ्गैराहुः तद्वच्च शोकतः। (A.H.Ni.8/12-13)

When the mind is agitated by fear (Bhaya), it can lead to a condition called Bhayaja Atisāra, in which both Pitta and Vāta are increased, causing the stool to become Drava (liquid). In the case of Atisāra, caused by Śoka (grief), the stool is Uṣṇa (warm) and Drava (liquid), and Plava (floats on water). The clinical features are similar to those of a combination of Vāta and Pitta Atisāra.

◉ तैस्तैर्भावैः शोचतोऽल्पाशनस्य बाष्पावेगः पक्तिमाविध्य(श्य)जन्तोः ॥ कोष्ठं गत्वा क्षोभयत्यस्य रक्तं तच्चाधस्तात् काकणन्तीप्रकाशम् । वर्चोमिश्रं निःपुरीषं सगन्धं निर्गन्धं वा सार्यते तेन कृच्छ्रात् ॥ शोकोत्पन्नो दुश्चिकित्स्योऽतिमात्रं रोगो वैद्यैः कष्ट एष प्रदिष्टः । (Su.U.40/13-15)

Due to Śocatoalpāśana (taking little food due to grief) and Bāṣpāvegaḥ (suppression of tears); the suppressed tears reaches the Koṣṭha and does Paktimāvidhya janto (afflicts the digestive process) and Kṣobayatyasya raktaṃ (excites the blood), which becomes Kākaṇantīprakāśam (dark red like Guñjā). Then Taccādhastāt (it passes down) through the rectum Varcomiśraṃ niḥpurīṣaṃ (mixed with faeces or without faeces) and Sagandhaṃ nirgandhaṃ (with or without odor with difficulty). This Śokaja Atisāra is considered as Duścikitsyoatimātraṃ (very hard to cure) and the physicians pointed out this as very troublesome.

Āmaja Atisāra

- आमाजीर्णोपद्रुताः क्षोभयन्तः कोष्ठं दोषाः सम्प्रदुष्टाः सभक्तम् ॥
  नानावर्णं नैकशः सारयन्ति कृच्छ्राज्जन्तोः षष्ठमेनं वदन्ति ॥ (Su.U.40/15-16)

Due to Āmājīrṇa (improper digestion of food), the doṣas in the Koṣṭha are aggravated and polluted, then expels the faeces repeatedly of Nānāvarṇa (various colours) mixed with undigested food with difficulty, it is known as sixth variety of Atisāra i.e. Āmaja Atisāra.

- संसृष्टमेभिर्दोषैस्तु न्यस्तमप्स्ववसीदति । पुरीषं भृशदुर्गन्धि विच्छिन्नं चामसञ्ज्ञकम् ।
  एतान्येव तु लिङ्गानि विपरीतानि यस्य तु । लाघवं च मनुष्यस्य तस्य पक्वं विनिर्दिशेत् ॥ (Su.U.40/17-18)

Saṃsṛṣṭamebhidoṣaistu (faeces mixed with the doṣas), Nyastamapsvavasīdati (sinks when dropped in water), Purīṣaṃ bhṛśadurgandhi (faeces emitting a very foul smell) and Vicchinnaṃ (broken faeces) are the clinical features of Āma Atisāra. Whereas, the features opposite to the above, especially Lāghavaṃ (lightness of the body) are the clinical features of Pakva Atisāra.

- तत्राद्ये गौरवादप्सु मज्जति। शकृद्दुर्गन्धमाटोपविष्टम्भार्तिप्रसेकिनः॥ विपरीतो निरामस्तु कफात्पक्वोऽपि मज्जति। (A.H.Ni.8/13-15)

In Sāmātisāra, the stool is described as Gaurava (heavy), Āpsu majjati (sinks in water), Durgandha (foul smell), accompanied by Āṭopa (gurgling sound), Viṣṭambha (stasis of undigested food in the stomach), Arti (pain in the abdomen), and Praseka (excessive salivation). The case of Nirāmātisāra (which does not have Āma) has opposite features, and the stool contains excessive Kapha and also sinks even when it is ripe.

Raktātisāra

- पित्तातिसारी यस्त्वेतां क्रियां मुक्त्वा निषेवते। पित्तलान्यन्नपानानि तस्य पित्तं महाबलम्॥
  कुर्याद्रक्तातिसारं तु रक्तमाशु प्रदूषयेत्। तृष्णां शूलं विदाहं च गुदपाकं च दारुणम्॥ (Ca.Ci.19/69-70)
- पित्तातिसारी यो मर्त्यः पित्तलान्यतिषेवते । पित्तं प्रदुष्टं तस्याशु रक्तातीसारमावहेत् ॥
  ज्वरं शूलं तृषां दाहं गुदपाकं च दारुणम् । (Su.U.40/116-117)

If the patient of Pittaja Atisāra does not follow the therapeutic measures and indulges in Pittalānyannapānā (the diet and drinks which increases the Pitta), then this Pitta become excessively aggravated and vitiates the Rakta immediately and produces the disease Raktātisāra associated with Jvara (fever), Tṛṣṇā (thirst), Śūla (colic), Dāha or Vidāha (burning sensation) and Gudapāka dāruṇa (severe anal suppuration).

Upadrava of Atisāra

- शूलप्रवाहिकाध्मानपरिकर्त्यरुचिज्वरान्। तृष्णोष्णदाहमूर्च्छादींश्चैषां विद्यादुपद्रवान्॥ (Ca.Si.8/22)

Śūla (colic), Pravāhika (tenesmus), Ādhmāna (abdominal distension), Parikartikā (sharp pain in the anal region), Aruci (anorexia), Jvara (fever), Tṛṣṇā (thirst), Uṣṇa (feeling of heat), Dāha (burning sensation) and Mūrcchā (fainting) are the complications of Atisāra

Sādhyāsādhyata of Atisāra

- पक्वजाम्बवसङ्काशं यकृत्खण्डनिभं तनु । घृततैलवसामज्जवेशवारपयोदधि ॥
  मांसधावनतोयाभं कृष्णं नीलारुणप्रभम् । मेचकं स्निग्धकर्बूरं चन्द्रकोपगतं घनम् ॥
  कुणपं मस्तुलुङ्गाभं सुगन्धि कुथितं बहु । तृष्णादाहतमःश्वासहिक्कापार्श्वास्थिशूलिनम् ॥
  सम्मूर्च्छारतिसम्मोहयुक्तं पक्ववलीगुदम् । प्रलापयुक्तं च भिषग्वर्जयेदतिसारिणम् ॥
  असंवृतगुदं क्षीणं दूराध्मातमुपद्रुतम् । गुदे पक्वे गतोष्माणमतिसारकिणं त्यजेत् ॥ (M.N.3/14-18)

If the faeces exhibits characteristics resembling the Pakvajambu (ripened fruit of Jambu) or Yakṛtkhaṇḍanibhaṃ (a piece of liver); and possesses a consistency akin to Ghṛta (ghee), Taila (oil), Vasā (fat), Majja (bone marrow), Veśavāra (meat preparation), Payas (milk), Dadhi (curd), Māṃsadhāvatoyābhaṃ (mutton wash); and appears Kṛṣṇa (black), Nīlāruṇaprabha (dark red colored), Mecaka (collyrium colored), with a Snigdha (greasy), Karbūra (variegated colour), Candragata (lustrous,) or Ghana (very thick), emitting an odor similar to that of Kuṇapaṃ (dead body), resembling Mastuluṅgā (brain contents), Sugandhi kuthota bahu (putrefied matter, or bearing a sweet smell,), and if the patient experiences symptoms such as Tṛṣṇa (thirst), Dāha (burning sensation), Tamaḥ (blackout), Śvāsa (dyspnoea), Hikkā (hiccups), Pārśvāsthi śūla (pain in the flanks and bones), Mūrccha (unconsciousness), Arati (restlessness), Moha (delusion), Pakvavalīguda (ulceration in the anus), and Pralāpa (delirium), it is advisable for a physician to refrain from treating such a patient. Furthermore, a wise physician should not accept a patient with diarrhoea if the elasticity of the anus is ulcerated and the body temperature decreases.

- श्वासशूलपिपासार्तं क्षीणं ज्वरनिपीडितम् । विशेषेण नरं वृद्धमतीसारो विनाशयेत् ॥ (M.N.3/19)

The patient with diarrhoea suffers simultaneously from Śvāsa (dyspnoea), Śūla (colic), Pipāsa (thirst), Kṣīṇa (emaciation) and Jvara (fever), and those Vṛddha (old aged) may die due to this disease Atisāra.

- (शोथं शूलं ज्वरं तृष्णां कासं श्वासमरोचकम् । छर्दिं मूर्छां च हिक्कां च दृष्टवातीसारिणं त्यजेत्) ।

If the complications of the disease produce Śotha (oedema), Śūla (colic), Jvara (fever), Tṛṣṇa (thirst), Kāsa (cough), Śvāsa (dyspnoea), Arocaka (anorexia), Chardi (vomiting), and Hikkā (hiccups), a wise physician should avoid the given case of Atisāra as the symptom is incurable.

Atisāra Mukti Lakṣaṇās / Nivṛtta Atisāra Lakṣaṇās

- ⊙ यस्योच्चारं विना मूत्रं सम्यग्वायुश्च गच्छति । दीप्ताग्नेर्लघुकोष्ठस्य स्थितस्तस्योदरामयः ॥ (Su.U.40/162)
- ⊙ यस्योच्चाराद्विना मूत्रं पवनो वा प्रवर्तते। दीप्ताग्नेर्लघुकोष्ठस्य शान्तस्तस्योदरामयः। (A.H.Ci.9/124)

Vinā mūtraṃ samyagvāyuśca gacchati (person is able to eliminate urine and flatus without eliminating the faeces), Dīptāgne (increased digestive fire) and Laghukoṣṭha (lightness in the abdomen) are the features which makes the disease Udarāmaya or Atisāra to be considered as cured.

In Contemporary science,

Atisāra can be correlated with diarrhoea in contemporary science. Diarrhoea is usually a symptom of GI tract infection caused by bacterial, viral and parasitic organism. WHO defined Diarrhoea as the passage of three or more loose or liquid stools per day (or more frequent passage than is normal for the individual) or is defined as the passage of frequent, liquid stools (usually of more than 200 g of stool) daily. Diarrhoea is broadly classified in to acute and chronic based on the duration.

Acute diarrhoea is defined as an episode lasting less than two weeks (within 14 days). Common causes include infections (viral, bacterial and parasite), food poisoning and medications (antibiotics, anti-neoplastics, etc.). Viral infection is the most common cause and the course is self-limited.

Chronic diarrhoea is defined as a duration lasting longer than two weeks (14 days or more) and tends to be non-infectious. Common causes include mal-absorption syndromes (celiac disease, carbohydrate intolerance especially lactose intolerance), irritable bowel syndrome, inflammatory bowel disease (crohn's disease and ulcerative colitis), endocrine disorders (hyperthyroidism and diabetes), colon carcinoma, etc.

Clinical features: The common presentations of diarrhoea include distension of the abdomen, abdominal discomfort/cramps/pain, nausea, vomiting, headache, loss of appetite and increased amounts of flatus which may be very foul-smelling, especially when digestion is impaired. In severe cases, signs of dehydration, bloody stool, fever, severe pain, and hypotension may develop. Psychiatric symptoms such as anxiety, loss of confidence, irritability and depression are very common, especially when diarrhoea is the prominent symptom.

Diagnosis: Typically in case of acute diarrhoea, lab testing or imaging studies are not equired as it has self-limited course. But in case of bloody stools or severe illness, stool culture s advised to rule out Shiga toxin, lactoferrin and Clostridium difficile toxin. CT abdomen may e required if there is presence of peritoneal signs. Blood investigations like complete blood count, basic metabolic panel, stimulating thyroid hormone, erythrocyte sedimentation rate, liver panel and stool analysis are the basic investigation in chronic diarrhoea. Sigmoidoscopy or colonoscopy with biopsies helps to identify the inflammatory causes.

PRAVĀHIKĀ

Introduction

Ācāryas described Pravāhikā in the same chapter of Atisāra because of the Atisāroktalakṣaṇamuktām (similar characteristic features) such as Purīṣa dravātāṃ (liquefication of the faecal material). Ācārya Ḍalhaṇa described that Pravāhikā is Atisāra bheda (types of Atisāra). Whereas Ācārya Āḍhamalla and Madhukoṣa differentiates the two terms viz. Atisāra and Pravāhikā, that there will be Nānādravadhātusaraṇa (expulsion of various watery dhātus through the anus) in Atisāra and Kevala Kaphasaraṇa (expulsion of only Kapha through the anus) in Pravāhika. Even Vācaspati in his Ātaṅkadarpaṇa opine the same that Pravāhikā is Śleṣmikapravāhayukta (expulsion of Kapha through the anus).

Nirūkti and Paribhāṣā

- ⊙ प्रवहति मुहुर्मुहुः प्रवर्त्तते इति । (SKD)

Pravāhikā is expulsion of faecal material repeatedly

- ⊙ प्रवाहमाणास्य भृशं सहामं विशोणितं वापि सशोणितं वा। स्वल्पं मुहुर्यस्य शकृन्निरेति प्रवाहका तस्य हि संप्रवृत्ता॥ (Si.Ni.5/286)

In which the person strains to defecate Alpa śakṛt (little quantity of faeces) with Āma frequently along with or without blood is called Pravāhikā.

- ⊙ अतिरूक्षेऽतिस्निग्धे वा भेषजमवचारितमप्राप्तं वातवर्च उदीरयति वेगाघातेन वा, तदा प्रवाहिका भवति; तत्र सवातं सदाहं सशूलं गुरु पिच्छिलं श्वेतं कृष्णं सरक्तं वा भृशं प्रवाहमाणः कफमुपविशति; तां परिस्रावविधानेनोपचरेत् ॥ (Su.Ci.34/18)

Ācārya Suśruta while describing the complications of emetic and purgative therapies, explained that the drug (purgative or emetic) administered to the person who is Atirūkṣa (excessive non-unctuous) or Atisnigdha (excessive unctuous) stimulates the impulse of Aprāpta Vātavarca (unmoved flatus and faeces) or by Vegāghātena (obstructing the natural urges) produces Pravāhika, in which he defecates the Kapha (mucous) which is Guru (heavy), Picchila (slimy), Śveta (white), Kṛṣṇa (black), Sarakta (mixed with blood) associated with Savāta (flatus), Sadāha (burning sensation) and Saśūla (pain)

Paryāya:

- प्रवाहिकैव भोजाद्यैर्विदांसीति निगद्यते। सैव निश्चारको नाम हारीतादिनये स्मृता॥ (Si.Ni.5/287)
- भोजादौ त्वियं विस्रंसीति नाम्ना पठ्यते, पराशर त्वन्तर्ग्रन्थिरिति, हारीतस्तु निश्चारकाख्यामेतां पठति। (Ma.M.N.3/21)
- विस्रांसी विस्रांसयति गुदभागं गुदभ्रंशादिना इति सेयं सञ्ज्ञा। निश्चारयति आमं रक्तं चेति निश्चारकः। पराशरमतेन तु अन्तर्ग्रन्ति संज्ञोऽयं रोग इति विजयरक्षितः। तत क्षतानां ग्रंन्थि सादृश्यात्॥ (Ga.Si.Ni.5/287)

The synonyms of Pravāhikā are Visraṃsī by Bhoja because it occurs as Visraṃsa (comes out) from Gudabhāga (anal route) leading to Gudabhraṃśa (ano-rectal prolapse) etc.; Nirçāraka by Harīta because it causes Nirçāraka (expulsion of) Āma and Rakta; and Antargranthi by Parāśara because it resembles Granthi caused by Kṣataṃ.

Sāmānya Nidāna and Saṃprāpti

- विबन्धादतिरूक्षोष्णा स्निग्धभक्ष्यनिषेवणात्। वेगसन्धारणाद् वापि विरेकव्यापदोऽपि वा॥
  विशेषाद् दुष्टजीवाणुसंक्रमाच्च जलादिना। प्रवाहिका प्रजायेत् स्वल्पा घोराऽथव नृणाम्॥ (Si.Ni.5/288-289)

Vibandha (constipation), Atirūkṣoṣṇā snigdha (intake of excessive dry, hot and unctuous substances), Vegasandhāraṇāt (suppression of natural urges), Virekavyāpat (complications of improper administration of purgative therapy) and specifically Duṣṭājīvāṇusaṃkrama jalādinā (invasion of micro-organism through water) are the causative factors of Pravāhikā.

- वायुः प्रवृद्धो निचितं बलासं नुदत्यधस्तादहिताशनस्य । प्रवाहमाणस्य मुहुर्मलाक्तं प्रवाहिकां तां प्रवदन्ति तज्ज्ञाः ॥ (Su.U.40/138)

Due to Ahitāśana (unwholesome diet), the Vāta gets aggravated and it carries down the *accumulated Balāsa* (mucous) into the lower part of the body, where it gets mixed with faeces *and it passed out frequently through the anus along with tenesmus; this disease is called Pravāhikā*

Saṃprāpti Ghaṭaka of Pravāhikā:

Doṣa	Kapha Pradhāna Tridoṣa
Dūṣya	Rasa
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa and Purīṣa vaha
Srotoduṣṭi	Atipravṛtti

Sañcāra sthāna	Koṣṭha
Udbhava sthāna	Āmapakvāśaya
Vyakta sthāna	Guda
Adhiṣṭhāna	Mahā srotas
Rogamārga	Abhyantara

Bheda and Lakṣaṇās of Pravāhikā

प्रवाहिका चतुर्था स्यात्पृथग्दोषैस्तथास्रतः ॥ (Śā.P.7/9)

Pravāhikā is of four types, one each from the doṣas viz., Vātaja, Pittaja, Kaphaja and the fourth one is from Raktaja.

- प्रवाहिका वातकृता सशूला पित्तात् सदाहा सकफा कफाच्च । सशोणिता शोणितसम्भवा तु ताः स्नेहरूक्षप्रभवा मतास्तु ॥ तासामतीसारवदादिशेच्च लिङ्गं क्रमं चामविपक्वतां च । (Su.U.40/139-140)

Vātaja Pravāhikā is associated with Śūlā (pain); Pittaja Pravāhikā is associated with Dāha (burning sensation); Kaphaja Pravāhikā is associated with Kaphā (mucus in the faeces) and Raktaja Pravāhikā is associated with Śoṇitasambhavā (presence of blood in the faeces). It is said that excessive use of Sneha (unctuous) and Rūkṣā (dry) food substances may be regarded as the exciting factor.

Pravāhikā Liṅga (clinical features), Krama (line of treatment) and Āmavipakvatāṃ (Āma and Pakva stages) are respectively identical with those of Atisāra.

Lakṣaṇa	Atisāra	Pravāhika
Svabhāva	Āśukārī	Āśukārī
Doṣa Pradhāna	Vāta Pradhāna Tridoṣa	Kapha Pradhāna Tridoṣa
Bheda	Six types	Four types
Pratyātma Lakṣaṇa	Atyanta sārayati mala	Sapravāhaṇa Muhurmuhur alpa śakṛt
Purīṣaja Lakṣaṇa	Nānā drava dhātu saraṇa	Kevala Kaphasaraṇa
Rakta darśana	Rakta may be found in faeces	Rakta is found in faeces
Āma doṣa	Sometimes Āma mala pravṛtti	Always Āma mala pravṛtti
Modern parlance	Diarrhoea	Dysentery

In Contemporary science,

Pravāhikā can be correlated with dysentery in contemporary science. Dysentery is an inflammatory condition of the intestine especially in the colon resulting in severe diarrhoea with mucus or blood in the faeces. It may be caused by bacterial, protozoal or parasitic infections and certain chemicals and it may spread to others through contaminated water and food products due to poor hygiene. The most common form of dysentery is shigellosis and amoebiasis.

Shigellosis (bacillary dysentery) is a food-borne or water-borne illness caused by gram-negative bacteria Shigella. There are four main groups namely Shigella dysenteriae, flexneri, boydii and sonnei. Among these, Shigella sonnei causes mild watery diarrhoea while Shigella flexneri and Shigella dysenteriae cause dysentery with bloody diarrhoea. The incubation period is 1-4 days and the mild cases resolve spontaneously within 4 – 8 days; whereas the severe cases take 3 - 6 weeks. Sometimes it may produce significant dehydration and electrolyte imbalance with circulatory collapse and death in debilitated patients.

Clinical features: It is often characterised by frequent small amounts of stools, and after a few evacuations it contain blood and pus with little faecal matter. Other symptoms include fever, nausea, vomiting, anorexia, lethargy, tenesmus and colicky abdominal pain. Fever and abdominal pain relieved by bowel movement may occur. In severe cases, symptoms like delirium, encephalopathy, seizures, meningismus and coma may occur.

Complication: Colon perforation, intestinal obstruction, toxic megacolon, rectal prolaps, haemolytic- uremic syndrome, reactive arthritis, iritis, keratitis and myocarditis.

Diagnosis: Diagnosis is based on the stool examination. Stool microscopic examination may show leucocytes and blood.

Amoebiasis (amoebic dysentery) is a parasitic infection caused by Entamoeba histolytica. There are three species of intestinal amoebas namely Entamoeba histolytica, Entamoeba dispar and Entamoeba moshkovskii. Among these, Entamoeba histolytica causes amoebic dysentery and amoebic liver abscesses. Amoebic infection occurs by the ingestion of mature cysts in contaminated food or water or from the hands. And also it may spread through sexually transmission by oral-anal contact. The incubation period from amoebiasis is between 1 to 4 weeks. However, the organism may remain in the bowel for many years without causing major symptoms.

Clinical features: The features are intermittent diarrhoea and constipation, mild abdominal pain especially right lower quadrant and stool may contain mucus and blood. The frequency ranges from 3 to 10 in a day. Sometimes bloody diarrhoea may occur. Other features include mild fever, lethargy, malaise, and weight loss.

Complication: Toxic megacolon, fulminant necrotizing colitis, colon perforation, Rectal prolapse, intussusception and colonic stricture

Diagnosis: Diagnosis is based on the stool examination, enzyme immunoassay of stool and serological testing. Stool microscopic examination may show leucocytes, erythrocytes and trophozoites and cysts of Entamoeba histolytica. Immunoassays that detect Entamoeba histolytica antigens in stool are sensitive and specific and are done to confirm the diagnosis.

Serological tests include enzyme-linked immunosorbent assay and polymerase chain reaction test to detect the antigen.

MCQ's

1. Atisāra is of .... types

a) 3
b) 4
c) 5
d) 6

2. According to Śārṅgadhara, Atisāra is of .... types

a) 5
b) 6
c) 7
d) 8

3. Ācārya Caraka in Siddhi sthāna described how many types of Atisāra

a) 24
b) 28
c) 32
d) 36

4. Pūrvarūpa of Atisāra is

a) Hṛdgudakoṣṭha toda
b) Gātra sāda
c) Mala graham
d) All of the above

5. Āmaja Atisāra is described by

a) Ācārya Caraka
b) Ācārya Vāgbhaṭa
c) Ācarya Suśruta
d) Both A and B

6. मांसतोयभ Atisāra is seen in

a) Vātaja Atisāra
b) Pittaja Atisāra
c) Kaphaja Atisāra
d) Sannipātaja Atisāra

7. Which condition is a common cause of chronic diarrhoea

a) Food poisoning
b) Medications
c) Viral infections
d) Irritable bowel syndrome

8. What are common clinical features of diarrhoea
   a) Joint pain and swelling
   b) Persistent cough
   c) Distension of the abdomen, abdominal pain, and nausea
   d) Skin rash and itching

9. Nānādravadhātusaraṇa is seen in
   a) Atisāra
   b) Pravāhikā
   c) Grahaṇī
   d) All of the above

10. Kevala Kaphasaraṇa is seen in
   a) Atisāra
   b) Pravāhikā
   c) Grahaṇī
   d) All of the above

11. According to Bhoja, Synonym of Pravāhikā is
   a) Visraṃsī
   b) Nirçāraka
   c) Antargranthi
   d) All of the above

12. Pravāhikā is of .... types
   a) 3
   b) 4
   c) 5
   d) 6

13. Which bacteria are common causes of bacillary dysentery
   a) Escherichia coli
   b) Shigella
   c) Salmonella typhi
   d) Staphylococcus aureus

14. What is the incubation period for shigellosis
   a) 1-4 hours
   b) 1-4 days
   c) 1-4 weeks
   d) 1-4 months

15. Which parasite causes amoebic dysentery
   a) Plasmodium falciparum
   b) Entamoeba histolytica
   c) Giardia lamblia
   d) Trichomonas vaginalis

Answer Keys

1	d	2	c	3	d	4	d	5	c	6	b	7	c	8	c
9	a	10	b	11	a	12	b	13	b	14	b	15	b		

Short Answer Questions

1. Explain Nidāna and Saṃprāpti of Atisāra
2. Differentiate the Doṣaja Atisāra
3. Explain Pravāhikā in detail
4. Write the difference between Atisāra and Pravāhikā

Long Answer Questions

1. Explain Nidāna, Saṃprāpti, bheda and Lakṣaṇās of Atisāra
2. Define and differentiate the Atisāra and Pravāhikā

◆

TOPIC 6

Grahaṇī

Introduction

Grahaṇī is an abode of Agni and its main function is Annasya grahaṇat (receiving the *food); hence it is named* Grahaṇī and is situated above the Nābhi (umbilicus) supported, *strengthened and nourished* by Agni. Ācārya Suśruta and Vāgbhaṭa described Grahaṇī as the *Ṣaṣṭī pittadharā kalā* situated between Āmāśaya (stomach) and Pakvāśaya (intestines). Usually, *Grahaṇī retains* the undigested food and releases the digested food from its left side. However, *if the Agni becomes* Durbala (weak), then it releases the Pakvāpakva (slightly digested and *undigested) food downwards* and this condition is called Grahaṇī gada or Grahaṇī roga or *Grahaṇī doṣa, which is* one among the Aṣṭamahā gada. Ācārya Caraka described Grahaṇī *after Arśas because Arśas is* a causative factor of Grahaṇīdoṣa. Ācārya Suśruta and Vāgbhaṭa *described Grahaṇī along with* Atisāra, because Grahaṇīroga occurs as a sequela of Atisāra.

Nirūkti and Paribhāṣā

- ⊙ ग्रहणीरोगमिति ग्रहण्या रोगो ग्रहणीरोगः। (Ma.M.N.4/3)

The disease which occurs in the Grahaṇī is known as Grahaṇīroga.

- ⊙ ग्रहणीमाश्रितोऽग्निदोषो ग्रहणीदोषः (Ca.Ca.Ci.15/1-2)

The Agnidoṣa (morbidity of Agni) located in the Grahaṇī is called Grahaṇīdoṣa.

- ⊙ अग्न्यधिष्ठानमन्नस्य ग्रहणाद्ग्रहणी मता। (Ca.Ci.15/56)
- ⊙ अग्न्यधिष्ठानमन्नस्य ग्रहणाद्ग्रहणी मता इति॥ (Āḍ.Śā.P.7/8)

Grahaṇī is an abode of *Agni* (digestive fire) which does Annasya grahaṇat (receives the food); *hence it is named* Grahaṇī.

- ⊙ अधस्तु पक्वमामं वा प्रवृत्तं ग्रहणीगदः। (Ca.Ci.15/52)

That which moves the Pakvamāmaṃ (slightly digested and undigested food substances) downwards into the Koṣṭha (gastrointestinal tract) is known as Grahaṇīgada.

Nidāna and Saṃprāpti

- ⊙ अतिसारे निवृत्तेऽपि मन्दाग्नेरहिताशिनः । भूयः सन्दूषितो वह्निर्ग्रहणीमभिदूषयेत् ॥ (Su.U.40/167)

If a person has Mandāgni even after subsidence of an attack of Atisāra and indulges in Ahitāśina (unwholesome food substances), his Agni (digestive power) further deteriorates and it leads to Grahaṇīmabhidūṣayet (affect the Grahaṇī).

- ⊙ अतीसारेषु यो नातियत्नवान् ग्रहणीगदः॥ तस्य स्यादग्निविध्वंसकरैरन्यस्य सेवितैः। (A.H.Ni.8/15-16)

If the patient of Atisāra, not making a proper effort to get cured of it, then he gets affected by Grahaṇīgada. And even others who indulge in Agnividhvaṃsaka (destructing the digestive fire) food and activities also gets affected by Grahaṇīgada.

- ⊙ यो हि भुङ्क्ते विधिं त्याक्त्वा ग्रहणीदोषजान् गदान्। स लौल्याल्लभते शीघ्रं,... । (Ca.Ci.15/41)

If the person consumes the food without following the Āhāra vidhi (rules of dietetics) due to Laulya (greediness), then he quickly suffers from the diseases developed by the vitiation of Grahaṇī.

- ⊙ एकशः सर्वशश्चैव दोषैरत्यर्थमुच्छ्रितैः । सा दुष्टा बहुशो भुक्तमाममेव विमुञ्चति ॥
  पक्वं वा सरुजं पूति मुहुर्बद्धं मुहुर्द्रवम् । ग्रहणीरोगमाहुस्तमायुर्वेदविदो जनाः ॥ (Su.U.40/171-172)

When the Grahaṇī gets deranged by the excessively aggravated doṣas either single or by combination, expels frequently the ingested food in the undigested or digested form, Muhurbaddha muhurdrava (sometimes constipated and sometimes diarrhoea) accompanied with Pūti (foul smell) and Śūla (pain in the abdomen). This condition is called as Grahaṇīroga by Ayurvedic scholars.

Saṃprāpti Ghaṭaka of Grahaṇī

Doṣa	Pitta Pradhāna Tridoṣa
Dūṣya	Rasa
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa, Anna and Purīṣa vaha
Srotoduṣṭi	Saṅga and Atipravṛtti
Sañcāra sthāna	Mahā srotas

Udbhava sthāna	Āmapakvāśaya
Vyakta sthāna	Grahaṇī
Adhiṣṭhāna	Grahaṇī
Rogamārga	Abhyantara

Pūrvarūpa of Grahaṇī

- पूर्वरूपं तु तस्येदं तृष्णाऽऽलस्यं बलक्षयः। विदाहोऽन्नस्य पाकश्च चिरात् कायस्य गौरवम्॥ (Ca.Ci.15/55)
- तस्योत्पत्तौ विदाहोऽन्ने सदनालस्यतृट्क्लमाः । बलक्षयोऽरुचिः कासः कर्णक्ष्वेडोऽन्त्रकूजनम् ॥ (Su.U.40/173)

Tṛṣṇā (thirst), Ālasya (malaise), Balakṣaya (diminution of strength), Vidāha (burning sensation), Cirātannasya pākā (delayed digestion of food) and Kāyasya Gaurava (heaviness of the body) are the premonitory symptoms of Grahaṇī. Ācārya Suśruta added Vidāhoanne (burning sensation after food), Sadana (lethargy), Klama (exhaustion), Aruci (anorexia), Kāsa (cough), Karṇakṣveḍa (tinnitus) and Antrakūjana (gurgling sound in the abdomen).

Sāmānya Lakṣaṇās of Grahaṇī

- अतिसृष्टं विबद्धं वा द्रवं तदुपदिश्यते। तृष्णारोचकवैरस्यप्रसेकतमकान्वितः॥
  शूनपादकरः सास्थिपर्वरुक् छर्दनं ज्वरः। लोहामगन्धिस्तिक्ताम्ल उद्गारश्चास्य जायते॥ (Ca.Ci.15/53-54)
- अथ जाते भवेज्जन्तुः शूनपादकरः कृशः । पर्वरुग्लौल्यतृट्छर्दिज्वरारोचकदाहवान् ॥
  उद्गिरेच्छुक्ततिक्ताम्ललोहधूमामगन्धिकम् । प्रसेकमुखवैरस्यतमकारुचिपीडितः ॥ (Su.U.40/174-175)

In Grahaṇī, the patient expels the faeces in Atisṛṣṭaṃ (excessive quantity), Vibaddhaṃ *vā drava (which may be solid or at times even liquid)* associated with Tṛṣṇā (thirst), Arocaka (anorexia), Āsyavairasya (distaste in the mouth), Praseka (salivation), Tamaka (feeling of darkness in front of eyes), Śūnapādakara (swelling in the hands and legs), Asthiparvaruk (pain in the bones and joints), Chardana (vomiting), Jvara (fever), Dāha (burning sensation), *Lohāmagandhi or Lohadhūmāmagandhika* (smell of iron, smoke and of undigested food in the breathe) and Tiktāmla udgāra (bitter or sour eructation).

Bheda of Grahaṇī

- स चतुर्धा पृथग्दोषैः सन्निपाताच्च जायते। (A.H.Ni.8/19)
- वातात् पित्तात् कफाच्च स्यात्तद्रोगस्त्रिभ्य एव च। (Ca.Ci.15/58)

Grahaṇī is classified into four types namely Vātaja, Pittaja, Kaphaja and Sannipātaja

- वातात् पित्तात् कफाच्चापि स रोगस्त्रितयादपि । सामवातेन चाप्यन्यः संग्रहग्रहणीगदः।
  सामश्लेष्मभ्वश्चान्यो राजग्रहणीकाह्वयः। जीर्णप्रवाहिकोत्थाऽन्या क्षतजा ग्रहणी तथा।
  क्षयता ग्रहणी चापि निर्मोकग्रहणी तथा। इत्येवं ग्रहणी रोग दृश्यन्ते बहुधा नृणाम्॥ (Si.Ni.5/340-341)

Ācārya Mādhavakara classified Grahaṇī into six types namely Vātaja, Pittaja, Kaphaja, Sannipātaja, Saṃgraha Grahaṇī and Ghaṭīyantra Grahaṇī.

Whereas Ācārya Gaṇanātha sen classified Grahaṇī into nine types namely Vātaja, Pittaja, Kaphaja, Sannipātaja, Saṃgraha Grahaṇī (produced by Sāma vāta), Rāja Grahaṇī (produced by Sāma Kapha), Kṣataja Grahaṇī (produced by Jīrṇapravāhika), Kṣayaja Grahaṇī (produced by Rājayakṣma) and Nirmoka Grahaṇī (produced by Āntrāntarakalāmaya (diseases of the inner layer of intestines)), thus Grahaṇīroga is of many kinds.

Bheda Lakṣaṇās

- वाताच्छूलाधिकैः पायुहृत्पार्श्वोदरमस्तकैः । पित्तात् सदाहैर्गुरुभिः कफात्त्रिभ्यस्त्रिलक्षणैः ॥
  दोषवर्णैर्नखैस्तद्वद्विण्मूत्रनयनाननैः । हृत्पाण्डूदरगुल्मार्शःप्लीहाशङ्खी च मानवः ॥ (Su.U.40/176-177)

In Vātaja Grahaṇī, there will be Śūlādhikaiḥ Pāyuhṛtpārśvodaramastakaiḥ (excessive pain in the anus, cardiac region, flanks, abdomen and head); Pittaja Grahaṇī is associated with Dāha (burning sensation); Kaphaja Grahaṇī is characterized with Gurutva (heaviness) and Tridoṣaja Grahaṇi exhibits all the symptoms of all the three doṣas.

The involved doṣas should be determined by the Varṇa (colour) of Nakha (nails), Viṇ (faeces), Mūtra (urine), Nayana (eyes) and Ānana (face). And the patient has a feeling that he is suffering from Hṛdroga (disease of the heart), Pāṇḍu (anaemia), Udara (ascites), Gulma, Arśas (piles), and Plīhā (splenomegaly).

Viśeṣa Nidāna, Saṃprāpti and Lakṣaṇās

Vātaja Grahaṇī

- कटुतिक्तकषायातिरूक्षशीतलभोजनैः। प्रमितानशनात्यध्ववेगनिग्रहमैथुनैः॥
  करोति कुपितो मन्दमग्निं सञ्छाद्य मारुतः । (Ca.Ci.15/59-60)

Due to excessive indulgence in Kaṭu (pungent), Tikta (bitter), Kaṣāya (astringent) food substances, Rūkṣa bhojana (non-unctuous food items), Śītala bhojana (excessive cold food items), Pramitāśana (eating less amount of food), Atyadhva (excessive travelling on foot), Veganigraha (suppression of natural urges) and Maithuna (sexual intercourse) causes the aggravation of Vāta which leads to Mandāgni (suppression of digestive fire) and produces the disease Vātaja Grahaṇī

- तस्यान्नं पच्यते दुःखं शुक्तपाकं खराङ्गता॥
  कण्ठास्यशोषः क्षुत्तृष्णा तिमिरं कर्णयोः स्वनः। पार्श्वोरुवङ्क्षणग्रीवारुजोऽभीक्ष्णं विसूचिका॥
  हृत्पीडा कार्श्यदौर्बल्यं वैरस्यं परिकर्तिका। गृद्धिः सर्वरसानां च मनसः सदनं तथा॥
  जीर्णे जीर्यति चाध्मानं भुक्ते स्वास्थ्यमुपैति च। स वातगुल्महृद्रोगप्लीहाशङ्की च मानवः॥
  चिरादुःखं द्रवं शुष्कं तन्वामं शब्दफेनवत्। पुनः पुनः सृजेद्वर्चः कासश्वासार्दितोऽनिलात्॥ (Ca.Ci.15/60-64)

The clinical features of Vātaja Grahaṇī are Anna pacyate duḥkhaṃ śuktapāka (food digested with difficulty and gets acidity), Kharāṅgatā (roughness in the body), Kaṇṭhāsya śoṣa (dryness of throat and mouth), Kṣut (excessive hunger), Tṛṣṇā (excessive thirst), Timira (blurred vision), Karṇayo svanaḥ (tinnitus), Pārśvoruvaṃkṣaṇagrīvārujoabhīkṣṇam (constant pain in the flanks, thighs, pelvis and neck), Visūcikā (pricking pain all over the body with vomiting and diarrhoea), Hṛtpīḍā (pain in the cardiac region), Kārśya (emaciation), Daurbalya (weakness), Vairasya (distaste in the mouth), Parikartikā (cutting pain), Gṛddhiḥ sarvarasānāṃ (longing for food of all tastes), Manasaḥ sadana (mental depression), Jīrṇe jīryati cādhmāna (distension of abdomen occurs during and after digestion), Bhukte svāsthyam (feels comfort immediately after intake of food) and the patient suspects as if he is suffering from Vātaja disorders, Gulma, Hṛdroga and Plīhā. The patient expels the faeces Punaḥpunaḥ (frequently) with Cirādduḥkha (difficulty after a long time) with Śabda (sound), and the faeces are Drava (liquid), Śuṣka (dried), Tanu (thin) associated with Āma (undigested food) and Phenavat (frothy). And also he may suffer from Kāsa (cough) and Śvāsa (dyspnoea).

Pittaja Grahaṇī

- ◉ कट्वजीर्णविदाह्यम्लक्षाराद्यैः पित्तमुल्वणम्। अग्निमाप्लावयद्धन्ति जलं तप्तमिवानलम्॥ (Ca.Ci.15/65)

Due to indulging in Kaṭu (pungent), Amla (sour), Kṣāra (alkali) food substances, Ajīrṇa (uncooked food) and Vidāhi (food causing burning sensation) etc., causes the aggravation of Pitta which leads to Agnimāplāvayaddhanti (extinguishes the digestive fire by drenching it) as hot water extinguishes the fire and produces the disease Pittaja Grahaṇī

- ◉ सोऽजीर्णं नीलपीताभं पीताभः सार्यते द्रवम्। पूत्यम्लोद्गारहृत्कण्ठदाहारुचितृडर्दितः॥ (Ca.Ci.15/66)

The clinical features of Pittaja Grahaṇī are Pūtyamlodgāra (sour eructation with foul *smell), Hṛtkaṇṭha dāha* (burning sensation in the cardiac and throat region), Aruci (anorexia), *Tṛḍ (thirst) and the patient* expels Drava (liquid) faeces containing Ajīr̄ṇa (undigested food *particles) with* Nīlapītābha (bluish- yellow) or Pītābha (yellow in colour).

Kaphaja Grahaṇī

- ◉ गुर्वतिस्निग्धशीतादिभोजनादतिभोजनात्। भुक्तमात्रस्य च स्वप्नाद्धन्त्यग्निं कुपितः कफः॥ (Ca.Ci.15/67)

Indulging in Guru bhojana (heavy food), Atisnigdha bhojana (excessive unctuous food), Śītādibhojana (cold food items etc.), Atibhojanāt (excessive intake of food) and Bhuktamātrasya *ca svapna (sleeping immediately after* intake of food) causes the aggravation of Kapha and *hampers the digestive fire simultaneously* and produces the disease Kaphaja Grahaṇī.

- ◉ तस्यान्नं पच्यते दुःखं हृल्लासच्छर्द्यरोचकाः। आस्योपदेहमाधुर्यकासष्ठीवनपीनसाः॥
  हृदयं मन्यते स्त्यानमुदरं स्तिमितं गुरु। दुष्टो मधुर उद्गारः सदनं स्त्रीष्वहर्षणम्॥
  भिन्नामश्लेष्म संसृष्टगुरुवर्चःप्रवर्तनम्। अकृशस्यापि दौर्बल्यमालस्यं च कफात्मके॥ (Ca.Ci.15/68-70)

The clinical features of *Kaphaja Grahaṇī are* Anna pacyate duḥkhaṃ (food is digested with difficulty), Hṛllāsa (nausea), Chardi (vomiting), Aruci (anorexia), Āsyopadeha (feeling as if mouth is coated), Mādhurya (sweet taste in the mouth), Kāsa (cough), Ṣṭhīvana (expectoration), Pīnasa (coryza), Hṛdayaṃmanyate (feels as if heart is stretched), Styānamudaraṃstimitaṃguru (idle, heavy abdomen and he feels as if the abdomen is covered with wet cloth), Duṣṭo madhura udgāra (sweet eructation with foul smelling), Sadana (lethargy), Strīṣvaharṣaṇa (absence of desire for woman) and the patient expels the Bhinnāmaśleṣmasaṃsṛṣṭaguru varca (fragmented faeces mixed with undigested, mucous and heavy) and Akṛśasyāpi daurbalyamālasya (he feels weakness and laziness even without emaciation).

Grahaṇī	Vātaja Grahaṇī	Pittaja Grahaṇī	Kaphaja Grahaṇī
Śārīrika Lakṣaṇa	Anna pacyate duḥkhaṃ śuktapāka, Kharāṅgatā, Kaṇṭhāsya śoṣa, Kṣut, Tṛṣṇā, Timira, Karṇayo svanaḥ, Pārśvoruvaṃkṣaṇagrīvārujoabhīkṣṇam, Visūcikā, Hṛtpīḍā, Kārśya, Daurbalya, Vairasya, Parikartikā, Gṛddhiḥ sarvarasānāṃ, Manasaḥ sadana, Kāsa and Śvāsa	Hṛtkaṇṭha dāha, Aruci, Tṛṣṇā	Anna pacyate duḥkhaṃ, Hṛllāsa, Chardi, Aruci, Āsyopadeha, Āsyamādhurya, Kāsa, Ṣṭhīvana, Pīnasa, Hṛdayaṃmanyate, Duṣṭo madhura udgāra, Sadana, Strīṣvaharṣaṇa, Akṛśasyāpi daurbalyamālasya
Purīṣaja Lakṣaṇa	Punaḥpunaḥ,Cirādduḥkha with Śabda, and the faeces are Drava, Śuṣka, Tanu associated with Āma and Phenavat	Sa Ajīrṇa drava mala, Nīlapītābha or Pītābha	Bhinnāma śleṣma saṃsṛṣṭa guru varca
Viśeṣa Lakṣaṇa	Jīrṇe jīryati cādhmāna, Bhukte svāsthyam	Pūtyamlodgāra	Styānamudaraṃ stimitaṃ guru

Sannipātaja Grahaṇī

- ◉ पृथग्वातादिनिर्दिष्टहेतुलिङ्गसमागमे। त्रिदोषं निर्दिशेत्तेषां.... । (Ca.Ci.15/72)

Hetu (aetiology) and Liṅga (clinical features) of Vātadi doṣas described separately will be appearing together in Sannipātaja Grahaṇī.

Saṃgraha Grahaṇī

- ◉ अन्त्रकूजनमालस्यं दौर्बल्यं सदनं तथा । द्रवं शीतं घनं स्निग्धं सकटीवेदनं शकृत् ।
  आमं बहु सपैच्छिल्यं सशब्दं मन्दवेदनम् । पक्षान्मासाद्दशाहाद्वा नित्यं वाऽप्यथ मुञ्चति ॥
  दिवा प्रकोपो भवति रात्रौ शान्तिं व्रजेच्च सा । दुर्विज्ञेया दुश्चिकित्स्या चिरकालानुबन्धिनी ॥
  सा भवेदामवातेन सङ्ग्रहग्रहणी मता । (M.N.4/17(1-3))

The clinical features of Saṃgraha Grahaṇī are Antrakūjana (gurgling sound in the abdomen), Ālasya (malaise), Daurbalya (weakness), Sadana (lassitude), Kaṭīvedana (pain in the waist) and the expelled faeces are Drava (watery), Śīta (cold), Ghana (solid), Snigdha (slimy), Āma (with undigested food materials), Bahu (copious), Sapaicchilya (sticky) associated with Saśabda (sound) and Mandavedana (mild pain). Such vitiated feaces are expelled once in fortnight or a month or in ten days or daily and the symptoms aggravate during day time and subside during the night. Such Cirakālanubadhinī (chronic) condition is caused by Āma and Vāta and it is Duścikitsya (difficult to treat).

Ghaṭīyantra Grahaṇī

- स्वपतः पार्श्वयोः शूलं गलज्जलघटीध्वनिः । तं वदन्ति घटीयन्त्रमसाध्यं ग्रहणीगदम् । (M.N.4/17(4))

The clinical features of Ghaṭīyantra Grahaṇī are Pārśvayo śūla (pain in the flanks) and Galajjalaghaṭīdhvani (produces sound in the abdomen resembling the water clock) and it is Asādhya (incurable).

Sādhyāsādhyata

- दोषं सामं निरामं च विद्यादत्रातिसारवत् ॥ लिङ्गैरसाध्यो ग्रहणीविकारो यैस्तैरतीसारगदो न सिध्येत् ।
  वृद्धस्य नूनं ग्रहणीविकारो हत्वा तनूं नैव निवर्तते च ॥ (M.N.4/18-19)

The Sāma and Nirāma avastha of doṣas and Asādhya liṅga (incurable features) in Grahaṇī are similar to those described in Atisāra. In Vṛddha (old aged), Grahaṇīroga is Asādhya (incurable) and it kills them.

- बालके ग्रहणी साध्या यूनि कृच्छ्रा समीरिता । वृद्धे त्वसाध्या विज्ञेया मतं धन्वन्तरेरिदम्) ॥ (M.N.4/19(1))

Lord Dhanvantari described that Grahaṇīroga is Sadhya (curable) in Bālaka (children), *Kṛcchra sādhya* (difficult to cure) in Yūni (young adults) and Asādhya (incurable) in Vṛddha *(old age).*

Upadravā

- अतीसारे ग्रहणयाञ्च दश कष्टा उपद्रवाः। ज्वरः शोथश्च शूलश्च भृशं हिक्का तथा वमिः।
  आध्मानं च प्रलापश्च तीव्रारोचक एव च। श्वासश्च पाण्डुता चेति सम्भवन्ति तु नैकधा॥ (Si.Ni.5/369-370)

Jvara (fever), *Śotha* (swelling), Śūla (colic), Bhṛśa hikka (frequent hiccups), Vami (vomiting), Ādhmāna (distension of abdomen), Pralāpa (delusion), Tīvrārocaka (severe anorexia), Śvāsa (dyspnoea) and Pāṇḍūtā (anaemia) are the ten complications of Atisāra and Grahaṇī. And these do not appear as one alone.

Difference between Grahaṇī Doṣa and Grahaṇī Roga

Grahaṇī Doṣa	Grahaṇī Roga
Preliminary stage of the Grahaṇī roga	Advanced stage and chronic condition of Grahaṇī doṣa
Grahaṇī doṣa refers to the vitiation of Agni, which is the functional derangement of Grahaṇī thāna.	When the vitiation of Agni within the Grahaṇī Sthāna results in structural deformity, the ensuing chronic condition is called as Grahaṇī Roga.

Grahaṇī Doṣa	Grahaṇī Roga
Due to Grahaṇī Āśritatva, Tikṣṇāgni, Mandāgni, Viṣamāgni can be considered as Grahaṇī doṣa, which is limited to the Āśaya (Grahaṇī).	Vitiated doṣas spreads throughout the body via Rasādhi dhātus i.e. Sarvaśārīra gata, causing Grahaṇī roga

Difference between Grahaṇī and Atisāra

	Atisāra	Grahaṇī
Svabhāva	Āśukārī	Cirakārī
Doṣa Pradhāna	Vāta Pradhāna Tridoṣa	Pitta Pradhāna Tridoṣa
Srotas	Purīṣavaha Srotas	Annavaha Srotas
Bheda	Six types	Four types
Sthāna	Pakvāśaya	Grahaṇī
Pratyātma lakṣaṇa	Atyanta sārayati mala	Muhurbaddha Muhurdrava mala pravṛtti
Purīṣaja lakṣaṇa	Nānā drava dhātu saraṇa	Pakvamāma vā pravṛtta
Modern parlance	Diarrhoea	IBS

In Contemporary science,

Grahaṇī can be predominantly characterised by conditions like irritable bowel syndrome (IBS) in contemporary science. (Please refer Topic 8).

Short Answer Questions

1. Define Grahaṇī and explain the Nidāna and Saṃprāpti
2. Differentiate the Doṣaja Grahaṇī
3. Write the difference between Grahaṇī doṣa and Grahaṇī roga
4. Write the difference between Atisāra and Grahaṇī
5. Explain Saṃgraha and Ghaṭīyantra Grahaṇī

Long Answer Questions

1. Explain Nidana, Saṃprāpti, Bheda, and Lakṣaṇās of Grahaṇī in detail

◆

TOPIC 7

Visūcikā, Alasaka, and Vilambikā

Introduction

- ⊙ अजीर्णमामं विष्टब्धं विदग्धं च यदीरितम् । विसूच्यलसकौ तस्माद्भवेच्चापि विलम्बिका ॥ (Su.U.56/3)
- ⊙ तं द्विविधमामप्रदोषमाचक्षते भिषजः- विसूचिकाम्, अलसकं च॥ (Ca.Vi.2/10)

Ācārya Suśruta described Alasaka, Visūcika and Vilambikā as the diseases occurring due *to three types of Ajīrṇa i.e.* Viṣṭabdhājīrṇa, Āmājīrṇa and Vidagdhājīrṇa respectively. Ācārya *Caraka described Alasaka* and Visūcikā as the types of Āmapradoṣaja vikāra in the Trividha *Kukṣīya Vimāna adhyāya.* Even Vāgbhaṭa opines the same as Caraka in Mātraśītīya adhyāya. *Ḍalhaṇa mentioned* that the disease Vilambikā is equated with the disease Daṇḍālasaka in *other texts.*

Sāmānya Nidāna and Saṃprāpti

- ⊙ न च खलु केवलमतिमात्रमेवाहारराशिमामप्रदोषकरमिच्छन्ति, अपि तु खलु गुरुरूक्षशीतशुष्कद्विष्टविष्टम्भिविदाह्यशुचिविरुद्धानामकाले चान्नपानानामुपसेवनं, कामक्रोधलोभमोहेर्ष्याह्रीशोक मानोद्वेगभयोपतप्तमनसा वा यदन्नपानमुपयुज्यते, तदप्याममेव प्रदूषयति॥ (Ca.Vi.2/8)

The causative factors for formation of Āma doṣas are Na ca khalu kevalamatimātrevāhāra (excessive intake of food is not only the cause of formation of Āma in the body), but also the use of diet and drinks which are Guru (heavy), Rūkṣa (non-unctuous), Śīta (cold), Śuṣka (dry), Dviṣṭa (disliked), Viṣṭambhi (constipating), Vidāhya (causing burning sensation in the abdomen), Aśuci (unclean), Viruddhā (contrary substances), Akāle (untimely) and the food taken when the mind is afflicted with Kāma (passion), Krodha (anger), Lobha (greed), Moha (delusion), Irṣyā (arrogance), Hrī (shame), Śoka (grief) and Bhaya (fear).

- ⊙ मात्रयाऽप्यभ्यवहृतं पथ्यं चान्नं न जीर्यति। चिन्ताशोकभयक्रोधदुःखशय्याप्रजागरैः॥ (Ca.Vi.2/9)

Even the most wholesome food taken in the proper quantity does not get digested, if the persons mind is afflicted with Cintā (worry), Śoka (grief), Bhaya (fear), Krodha (anger), Duḥkhaśayyā (uncomfortable bed) and Prajāgaraiḥ (keeping awake during the night time).

By these aforesaid factors, the three Doṣas gets aggravated which impairs the Agni (digestive fire) and leads to Agnimāndhya resulting in production of Āma doṣa which inturn causes Alasaka, Visūcikā and Vilambīkā.

- ⊙ अतिमात्रं त्वशनं त्रीनपि दोषान् प्रकोपयेत्। पीड्यमाना हि वाताद्या युगपत्तेन कोपिताः॥
  आमेनान्नेन दिष्टेन तदेवाविश्य कुर्वते। विष्टम्भयन्तोऽलसकं च्यावयन्तो विसूचिकाम्॥
  अधरोत्तरमार्गाभ्यां सहसैवाजितात्मनः । (A.H.Sū.8/4-6)

Excessive intake of food causes the sudden vitiation of Vātadi doṣas and Āmā along with Duṣṭānna (vitiated food substances) further vitiates them and together produces the disease Alasaka by Viṣṭambha (blocking the movement of food) and Visūcikā by Adharottaramārgabhyāṃ sahasai cyāvayati (moving in downward and upward directions simultaneously) in person who is Ajitātmanaḥ (not self controlled).

Visūcikā

Nirūkti and Paribhāṣā

- ⊙ विविधसूचीतुल्यत्वाद्विसूचिका। (He.A.H.Sū.8/7-8)

That which produces different types of pain similar to pricking with needle is known as Visūcikā.

- ⊙ विविधानां विकाराणां सूचिका-विसूचिकेत्युच्यते। (Ar.A.H.Sū.8/7-8)

That which produces different types of pricking pain is known as Visūcikā

- ⊙ यस्माद्गात्राणि सूचिभिरिव विध्यति, तस्माद्विसूचिकेत्युच्यते। (Ar.A.H.Sū.8/7-8)

That which produces pain all over the body as though being pricked by needles is known as Visūcikā

- ⊙ विशेषण सूचीभिरिव मर्माणि पीडयतीति विसूची॥ (Āḍ.Śā.P.7/11)

That which produces pricking pain in the Marmāṇi (vital parts of the body) is known as Visūcikā.

- ⊙ बाहुल्याद्वायुः सूचीभिरिव तुदन्' इति विसूचीनिरुक्तिः (Ma.M.N.6/16-17)

That which produces pricking type of pain due to aggravation of Vāta is known as Visūcikā.

- ⊙ विविधैर्वेदनोद्भेदैर्वाय्वादिभृशकोपतः॥ सूचीभिरिव गात्राणि विध्यतीति विसूचिका। (A.H.Sū.8/7-8)

Aggravation of Vātadi doṣas causing different types of pain as though being pricked by needles; therefore it is called as Visūcikā.

- सूचीभिरिव गात्राणि तुदन् सन्तिष्ठतेऽनिलः । यस्याजीर्णेन सा वैद्यैरुच्यते ति विसूचिका ॥ (Su.U.56/4)

If the patient of Ajīrṇa suffering from Sūcībhiriva gātrāṇi (pricking pain in the body) due to the vitiation of Vāta, then the condition is called as Visūcīka by the physicians.

Nidāna and Saṃprāpti

- न तां परिमिताहारा लभन्ते विदितागमाः । मूढास्तामजितात्मानो लभन्ते कलुषाशयाः (Su.U.56/5)

The Vidita (learned person) with the knowledge about the Parimitāhārā (moderation in diet) may not possess this disease, only the Mūḍha (ignorant person) with Ajitātmāno (Ajitendriya – having subdued digestive fire) develops Kaluṣāśayāḥ (Āmāśaya duṣṭi) which in turn produces the disease Visūcikā.

Saṃprāpti Ghaṭaka

Doṣa	Tridoṣa
Dūṣya	Rasa
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa, Anna and Purīṣa vaha
Srotoduṣṭi	Saṅga, Atipravṛtti and Vimārga gamana
Sañcāra sthāna	Mahā srotas
Udbhava sthāna	Āmāśaya
Vyakta sthāna	Sarva śārīra
Adhiṣṭhāna	Āmāśaya
Rogamārga	Abhyantara

Lakṣaṇās

- मूर्च्छातिसारौ वमथुः पिपासा शूलं भ्रमोद्वेष्टनजृम्भदाहाः । वैवर्ण्यकम्पौ हृदये रुजश्च भवन्ति तस्यां शिरसश्च भेदः ॥ (Su.U.56/6)

Mūrcchā (fainting), Atīsāra (diarrhoea), Vamathu (vomiting), Pipāsā (thirst), Śūla (colic), rama (giddiness), Udveṣṭāna (convulsions), Jṛmbha (yawning), Dāha (burning sensation),

Vaivarṇya (discoloration of the body), Kampa (tremors), Hṛdaye ruja (pain in the cardiac region) and Śirasa bheda (headache) are the features of Visūcikā.

- तत्र विसूचिकामूर्ध्वं चाधश्च प्रवृत्तामदोषां .... । (Ca.Vi.2/11)

Whereas Ācārya Caraka described that the Āmadoṣa expelled out from upper (vomiting) and lower (diarrhoea) passages is known as Visūcīka.

- तत्र वातः शूलानाहाङ्गमर्दमुखशोषमूर्च्छाभ्रमाग्निवैषम्य- पार्श्वपृष्ठकटिग्रहसिराकुञ्चनस्तम्भनानि करोति, पित्तं पुनर्ज्वरातीसारान्तर्दाहतृष्णामदभ्रमप्रलपनानि, श्लेष्मा तु छर्द्यरोचकाविपाकशीतज्वरालस्यगात्रगौरवाणि॥ (Ca.Vi.2/7)

Śūla (colic), Ānāha (abdominal distension with constipation), Aṅgamarda (body pain), Mukhaśoṣa (dryness of the mouth), Mūrcchā (fainting), Bhrama (giddiness), Agnivaiṣamya (irregularity in the digestive fire), Pārśvapṛṣṭhakaṭigraha (stiffness of the flanks, back and waist), Sirākuñcanastambhanāni (contraction and spasms of blood vessels) are the features of **Vātaja Visūcikā.**

Pittaja Visūcikā produces Jvara (fever), Atīsāra (diarrhoea), Antardāha (burning sensation inside the body), Tṛṣṇā (thirst), Mada (intoxication), Bhrama (giddiness) and Pralapanāni (delirium);

Kaphaja Visūcikā causes Chardi (vomiting), Arocaka (anorexia), Avipāka (indigestion), Śītajvara (fever with cold), Ālasya (laziness) and Gātragauravāṇi (heaviness of body).

Upadrava

- निद्रानाशोऽरतिः कम्पो मूत्राघातो विसञ्ज्ञता । अमी ह्युपद्रवा घोरा विसूच्यां पञ्च दारुणाः ॥ (M.N.6/25)

Nidranāśa (loss of sleep), Arati (restlessness), Kampa (tremors), Mūtraghāta (retention of urine) and Visaṃjña (unconsciousness) are the complications of Visūcikā.

Sādhyāsādhyata

- यः श्यावदन्तौष्ठनखोऽल्पसञ्ज्ञश्छर्द्यर्दितोऽभ्यन्तरयातनेत्रः । क्षामस्वरः सर्वविमुक्तसन्धिर्यायान्नरः सोऽपुनरागमाय ॥ (Su.U.56/11)

Śyāvadantauṣṭhanakha (blackish blue discolouration of teeth, lips and nails), Alpasaṃjña (diminished consciousness), Chardi (vomiting), Ābhyantarayātanetra (Kṣīṇa dṛṣṭi – diminished vision), Kṣāmasvara (feeble voice) and Sarvavimuktasandhi (looseness of all the joints), should be considered that the disease reached an incurable stage with no hope of recovery.

ALASAKA

Nirūkti and Paribhāṣā

- अलसत्वं-निष्क्रितत्वम्।(He.A.H.Sū.8/6-7)

The term Alasa means *Niṣkriyatva* (inactive)

- दोषस्थिरत्वनिमित्तादलसक संज्ञा॥ (*Āḍ.Śā.P.7/10*)
- 'अलसक' इति दोषस्थिरत्वनिमित्ता सञ्ज्ञा ।(Ma.M.N.6/19-20)

That which causes *Doṣasthiratva* (immovability of the doṣas) is called as Alasaka.

- कोष्ठेस्थितोऽलसीभूतस्तेन चालसकः समृतः॥ (Āḍ.Śā.P.7/10)

That which causes ingested food to remain idle in the Koṣṭha (stomach) is called Alasaka

- प्रयाति नोर्ध्वं नाधस्तादाहारो न च पच्यते॥ आमाशयेऽलसीभूतस्तेन सोऽलसकः स्मृतः। (A.H.Sū.8/6-7)

The condition where the ingested food remains idle in the stomach without undergoing digestion and neither moves upwards (vomiting) nor downwards (purgation) is known as *Alasaka.*

Saṃprāpti Ghaṭaka

Doṣa	Vātakapha Pradhāna Tridoṣa
Dūṣya	Rasa
Agni	*Jaṭharāgni māndhya*
Āma	*Jaṭharāgni māndhya janya Āma*
Srotas	Rasa and Anna vaha
Srotoduṣṭi	*Saṅga*
Sañcāra sthāna	*Mahā srotas*
Udbhava sthāna	*Āmāśaya*
Vyakta sthāna	*Āmāśaya*
Adhiṣṭhāna	Āmāśaya
Rogamārga	Abhyantara

Saṃprāpti and Lakṣaṇās

- अलसकमुपदेक्ष्यामः- दुर्बलस्याल्पाग्नेर्बहुश्लेष्मणो वातमूत्रपुरीषवेगविधारिणः स्थिरगुरुबहुरूक्षशीतशुष्कान्नसेवि नस्तदन्नपानमनिलप्रपीडितं श्लेष्मणा च विबद्धमार्गमतिमात्रप्रलीनमलसत्वान्न बहिर्मुखीभवति, ततश्छर्दतीसार वर्ज्यान्यामप्रदोष लिङ्गान्यभिदर्शयत्यतिमात्राणि। (Ca.Vi.2/12)

Alasaka occurs especially in those who are Durbala (weak person), having Alpāgni (weak digestive power) and Bahuśleṣma (abundance of Kapha in the body), Vātamutrapurīṣavegavirodhiṇa (suppresses the urges of passing flatus, urine and faeces), Sthiragurubahurūkṣaśītaśūṣkānnasevina (excessively consumes solid, heavy, excessively non- unctuous, cold and dry foods), Tadannapānamanila prapīḍitaṃ (such diet and drinks gets afflicted with Vāta). Śleṣmaṇa ca vibaddha mārgamatimātra pralīna malasatvānnabahirmukhībhavati (the waste products does not move to external orifices due to the obstruction of passage by Kapha and excessive adhesiveness of the undigested food). And it produces all the clinical features of Āmadoṣa except Chardi (vomiting) and Atīsāra (diarrhoea).

- कुक्षिरानह्यतेऽत्यर्थं प्रताम्येत् परिकूजति । निरुद्धो मारुतश्चैव कुक्षावुपरि धावति ॥
  वातवर्चोनिरोधश्च यस्यात्यर्थं भवेदपि । तस्यालसकमाचष्टे तृष्णोद्गारौ च यस्य तु ॥ (Ma.M.N.6/19-20)
- कुक्षिरानह्यतेऽत्यर्थं प्रताम्यति विकूजति । निरुद्धो मारुतश्चापि कुक्षौ विपरिधावति ॥
  वातवर्चोनिरोधश्च कुक्षौ यस्य भृशं भवेत् । तस्यालसकमाचष्टे तृष्णोद्गारावरोधकौ ॥ (Su.U.56/7-8)

Kukṣirānahyateatyarthaṃ (excessive distension of abdomen), Pratāmyati (feeling of entering in the dark), Vikūjati (rumbling sound in the intestines), Niruddho mārutaścāpi kukṣau viparidhāvati (Vāyu moves all around the abdomen due to obstruction of Vāta), Vātavarconirodha (absolute obstruction of faeces and flatus), Tṛṣṇodgārāvarodhaka (hinders the hiccups and eructation) are the clinical features of Alasaka.

Daṇḍālasaka

- तच्चालसकभेदभिन्नलक्षणत्वेन पृथग्दर्शितम्। तद्यथा – तिर्यग्गतास्तनुं दोषा दण्डवत्।
  स्तम्भयन्ति ये। स दण्डालसकस्त्याज्यः शीघ्रं देहविनाशकृत् इति॥ (Āḍ.Śā.P.7/11)

Daṇḍālasaka is a type of Alasaka which manifests with different features i.e. the doṣas moves sideward and makes the body as Daṇḍavat stambhayanti (stiff like a wooden shaft) and destructs the body quickly. Hence it should be rejected from the treatment.

- अतिमात्रप्रदुष्टाश्च दोषाः प्रदुष्टामबद्धमार्गास्तिर्यग्गच्छन्तः कदाचिदेव केवलमस्य शरीरं दण्डवत् स्तम्भयन्ति, ततस्तं दण्डालसकमसाध्यं ब्रुवते। (Ca.Vi.2/12)

- अत्यर्थदुष्टास्तु दोषा दुष्टामबद्धखाः। यान्तस्तिर्यक्तनुं सर्वां दण्डवत्स्तम्भयन्ति चेत्॥
  दण्डकालसकं नाम तं त्यजेदाशुकारिणम्। (A.H.Sū.8/12-13)

Excessively vitiated doṣas moves obliquely due to the obstruction of passage by Āmadoṣa, which in turn causes Śarīraṃ daṇḍavat stambhayanti (stiffening of the body like a wooden shaft, this disease is known as Daṇḍālasaka or Daṇḍakālasaka, which is Asādhya (incurable).

- विरुद्धाध्यशनाजीर्णाशनशीलिनः पुनरामदोषमामविषमित्याचक्षते भिषजः, विषसदृशलिङ्गत्वात्; तत् परमसाध्यम्, आशुकारित्वाद्विरुद्धोपक्रमत्वाच्चेति॥ (Ca.Vi.2/12)

Āmadoṣa of such persons who regularly consumes Viruddha (contrary food substances) Adhyāśana (eating before digestion of previous food) and Ajīrṇāśīlinaḥ (improperly cooked food substances) is called Āmaviṣa by physicians because it manifests the features like that of poison. It is Paramasādhya (absolutely incurable) because of its Āśukārītva (acuteness) and Viruddhopakrama (contradictory line of management).

VILAMBIKĀ

Nirūkti and Paribhāṣā

- दीर्घकालविलम्बनात् विलम्बिकेत्युच्यते। (He.A.H.Sū.8/28)

The term Vilambika refers Vilamba (delay) and Dīrghakāla (long term).

- विसूच्यलसकौ तस्माद्भवेच्चापि विलम्बिका इति॥ (Āḍ.Śā.P.7/11)

That which occurs with the features of Visūcika and Alasaka is known as Vilambikā

- गरीयसो भवेल्लीनादामादेव विलम्बिका। (A.H.Sū.8/28)

The disease Vilambikā is due to excessive accumulation of Āma within the Srotomārgas in the latent form.

Samprāpti Ghaṭaka

Doṣa	Kaphavāta Pradhāna Tridoṣa
Dūṣya	Rasa
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa and Anna vaha
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Mahā srotas
Udbhava sthāna	Āmāśaya
Vyakta sthāna	Sarva śārīra
Adhiṣṭhāna	Āmāśaya
Rogamārga	Abhyantara

Lakṣaṇās

- दुष्टं तु भुक्तं कफमारुताभ्यां प्रवर्तते नोर्ध्वमधश्च यस्य । विलम्बिकां तां भृशदुश्चिकित्स्यामाचक्षते शास्त्रविदः पुराणाः ॥ (Su.U.56/9)

The condition where the ingested food becomes polluted with Kapha and Vāta and does not move neither upwards nor downwards is known as Vilambikā by the ancient physicians and it is Duścikitsya (difficult to treat). Ḍalhaṇa while commenting on this mentioned that the disease Vilambikā is equated with the disease Daṇḍālasaka in other texts.

In Contemporary science,

Alasaka, Visūcikā and Vilambikā are purely Āyurvedic concept and it cannot be correlated with any specific disease in contemporary science. Some scholars attempted to correlate Visūcikā with Gastroenteritis; Alasaka and Vilambikā with Gastric atony in modern science.

Gastroenteritis

Gastroenteritis is characterized by inflammation of the mucosal lining of the stomach and both the small and large intestines. It is frequently attributed to infections caused by various pathogens, including viruses such as norovirus, rotavirus, astrovirus, and adenoviruses; bacteria including Campylobacter, Clostridioides difficile, Escherichia coli, Salmonella, Shigella, and Staphylococcus; as well as parasites like Giardia and Cryptosporidium parvum. Additionally, gastroenteritis may result from foodborne illnesses and certain pharmacological agents, including antibiotics and non-steroidal anti-inflammatory drugs (NSAIDs).

Globally, Gastroenteritis ranks as the third leading cause of death among children under five years of age, accounting for 15% of all fatalities in this demographic. The principal risk factors encompass consumption of contaminated food and water, poor hygiene practices, and inadequate sanitation.

Clinical features: The common clinical features include diarrhoea, often watery or bloody, and vomiting. Patients often report abdominal cramps, tenderness, and bloating. Additionally, low-grade fever and general malaise are prevalent. Nausea and loss of appetite may precede the onset of other symptoms. In certain cases, patients may experience headaches, chills, and generalized body aches. The severity of the illness can fluctuate, ranging from a mild, self-limiting condition to severe dehydration, which poses a significant risk, particularly in vulnerable populations such as the elderly, young children, and individuals with compromised immune systems. Additional symptoms may encompass weight loss, flatulence, and rectal urgency.

Complications: This condition may lead to various complications, including dehydration, electrolyte imbalances, hypovolemic shock, septicemia, renal failure, post-infectious irritable bowel syndrome, inflammatory bowel disease, Guillain-Barre syndrome, reactive arthritis, and colon perforation.

Diagnosis: The diagnosis of gastroenteritis is primarily based on clinical evaluation complemented by stool examination. Stool microscopy, culture, and sensitivity testing are instrumental in identifying specific infectious agents. In certain cases, additional diagnostic tools, including endoscopy, imaging studies, or biomarkers, may be utilized.

Gastric Atony

Gastric atony, also known as Gastroparesis, is a condition characterized by delayed gastric emptying. This condition arises from weakened or abnormal muscle contractions within the stomach, occurring in the absence of any mechanical obstruction, and persists for duration of at least three months. The global prevalence of Gastroparesis is estimated to be approximately 2% of the population, with a higher incidence reported in women compared to men; however, the underlying reasons for this gender disparity remain unclear. The majority of cases of Gastroparesis are classified as idiopathic. Notable risk factors include diabetes mellitus, complications following surgical procedures, and the use of certain medications.

Clinical features: The most prevalent clinical symptoms of this condition are nausea and vomiting, which correspond with the extent of gastric emptying delay. Other associated symptoms may include abdominal bloating, pain and discomfort, early satiety, postprandial fullness, weight loss, regurgitation of food, and dyspepsia.

Complications: Potential complications associated with gastric atony include severe malnutrition, dehydration, electrolyte imbalances, an increased risk of infections, the formation of bezoars, and Mallory-Weiss tears.

Diagnosis: Diagnosis of gastric atony is based on clinical evaluation, laboratory tests, and imaging studies. Patients frequently present with symptoms such as nausea, vomiting, abdominal bloating, and early satiety. Physical examinations may reveal abdominal distension and tenderness. Diagnostic assessments may include upper gastrointestinal (GI) endoscopy to exclude mechanical obstruction, alongside gastric emptying studies utilizing scintigraphy or gastric emptying breath tests to evaluate the rate of gastric emptying. Electrogastrography (EGG) may also be employed to assess gastric electrical activity. Further evaluations, such as gastric manometry, may be necessary to comprehensively assess gastric motility. A definitive diagnosis is established when gastric emptying studies indicate delayed gastric emptying, typically defined as the retention of more than 10% of the meal at four hours or more than 5% at six hours.

MCQ's

1. According to Caraka, Āmapradoṣaja Vikāras are

a) Alasaka b) Visūcikā
c) Vilambikā d) Both a and b

2. Āmapradoṣaja Vikāra is of _ types

a) 2 b) 3
c) 4 d) 5

3. Alasaka is manifested due to

a) Viṣṭabdhājīrṇa b) Āmājīrṇa
c) Vidagdhājīrṇa d) Dinapāki ajīrṇa

4. Visūcikā is

a) Āmadoṣa expelled out from upper and lower passages
b) Āmadoṣa remains idle in the stomach
c) Excessive accumulation of Āma within the Srotomārga in the latent form
d) None of the above

5. According to Caraka, Visūcikā is of .... types

a) 3 b) 5
c) 7 d) 9

6. According to Mādhavakara, Visūcikā has .... Upadravas

a) 3 b) 5
c) 7 d) 9

7. Alasaka is
   a) Āmadoṣa expelled out from upper and lower passages
   b) Āmadoṣa remains idle in the stomach
   c) Excessive accumulation of Āma within the Srotomārga in the latent form
   d) None of the above
8. Vilambikā is
   a) Āmadoṣa expelled out from upper and lower passages
   b) Āmadoṣa remains idle in the stomach
   c) Excessive accumulation of Āma within the Srotomārga in the latent form
   d) None of the above
9. What characterizes gastroenteritis
   a) Inflammation of the lungs
   b) Inflammation of the mucosal lining of the stomach and intestines
   c) Inflammation of the joints
   d) Inflammation of the liver
10. What are common clinical features of gastroenteritis
   a) Chronic cough and wheezing
   b) Diarrhoea, vomiting, and abdominal cramps
   c) Joint pain and swelling
   d) Persistent rash and itching
11. What are potential complications of gastroenteritis
   a) Dehydration and electrolyte imbalances
   b) Hypovolemic shock and septicemia
   c) Renal failure and Guillain-Barre syndrome
   d) All of the above

What characterizes gastric atony (gastroparesis)?
a) Rapid gastric emptying
b) Delayed gastric emptying
c) Normal gastric emptying
d) Increased gastric motility

13. What are common risk factors for gastroparesis?
   a) High-fat diet
   b) Diabetes mellitus
   c) Lack of exercise
   d) Sedentary lifestyle
14. Which clinical symptoms are most prevalent in gastric atony?
   a) Chronic cough and wheezing
   b) Nausea and vomiting
   c) Joint pain and swelling
   d) Skin rash and itching

Answer Keys

1	d	2	a	3	a	4	a	5	a	6	b	7	b
8	c	9	b	10	b	11	d	12	b	13	b	14	b

Short Answer Questions

1. Explain Visūcikā
2. Explain Alasaka
3. Explain Vilambikā

◆

TOPIC 8

Common Gastrointestinal Tract Diseases

Introduction

The gastrointestinal (GI) tract, also known as the digestive system, is a complex and essential organ system responsible for the processes of consumption, digestion, absorption, and elimination of food and waste products. This intricate system comprises several components, including the mouth, oesophagus, stomach, small intestine, large intestine (colon), rectum, and anus.

A range of diseases and disorders can compromise the functionality of the gastrointestinal (GI) tract, affecting millions of individuals globally. Among the most prevalent GI tract conditions are Gastroesophageal Reflux Disease (GERD), Irritable Bowel Syndrome (IBS), Inflammatory Bowel Disease (IBD), Peptic Ulcer Disease, Celiac Disease, Diverticulitis, Gastrointestinal Cancer, Hemorrhoids, Gallstones, Hepatitis, Cirrhosis, and Pancreatitis. This chapter will provide a comprehensive analysis of specific conditions, including dyspepsia, irritable bowel syndrome and inflammatory bowel diseases.

DYSPEPSIA

Introduction

Dyspepsia, also known as indigestion, is a condition characterized by recurrent or persistent pain or discomfort in the upper abdomen. It encompasses symptoms such as bloating, pain, postprandial fullness, heartburn and belching. The global occurrence of dyspepsia is 20-30%, with a slightly higher prevalence in the Western population and a higher frequency among women. While the exact prevalence of dyspepsia in India is not known, various studies estimate that it affects 7.6-49% of the Indian population.

Causes of dyspepsia include Helicobacter pylori gastritis, gallstones, irritable bowel syndrome, oesophageal spasm, Gastroesophageal reflux disease, pancreatic disease, hepatic disease, renal failure, hypercalcemia, anxiety, depression, smoking, alcohol consumption, and the use of certain drugs such as NSAIDs, glucocorticoids, digoxin, iron, and potassium supplements.

Pathophysiology

Dyspepsia is categorized as ulcer dyspepsia or non-ulcer dyspepsia (functional dyspepsia).

Ulcer dyspepsia refers to indigestion caused by peptic ulcer disease (PUD), encompassing gastric and duodenal ulcers. The principal factors contributing to the pathophysiology of ulcer dyspepsia are Helicobacter pylori Infection, Non-steroidal Anti-Inflammatory Drugs (NSAIDs), Gastric Acid Secretion and Life style factors such as Smoking, alcohol consumption, and stress. Ulcer dyspepsia can cause pain or discomfort in the upper abdomen, bloating, belching, and nausea.

Non-ulcer dyspepsia, also known as functional dyspepsia, is delineated by the revised Rome IV criteria, which encompass the following:

- Persistent or recurring dyspepsia for more than 3 months within the past 6 months
- No demonstration of a possible organic cause of the symptoms on endoscopy
- No sign that the dyspepsia is relieved only by defecation or of an association with stool irregularities

This final criterion was implemented to exclude irritable bowel syndrome (IBS) as a potential factor for the symptoms. However, it is worth noting that approximately 30% of patients diagnosed with functional dyspepsia also exhibit symptoms of IBS. Under the current Rome IV criteria, functional dyspepsia is categorized into two subgroups based on the predominant symptoms:

1. **Epigastric Pain Syndrome (EPS)** - characterized by predominant epigastric pain or burning
2. **Postprandial Distress Syndrome (PDS)** - characterized by a sensation of fullness and early satiety.

Clinical features:

The common features are epigastric burning pain, abdominal discomfort, anorexia, belching, constipation, halitosis, chronic cough or sore throats, diaphoresis, nausea and vomiting. Alarm features are hematemesis, anaemia, dysphagia, palpable abdominal mass, weight loss and malena.

Diagnosis:

Ulcer dyspepsia is diagnosed based on the lab investigations and endoscopy. Functional dyspepsia is diagnosed based on the typical symptoms and the patient's history; and the exclusion of other diseases of the upper gastrointestinal tract and upper abdominal organs that may present with similar dyspeptic symptoms.

The diagnostic examinations deemed sufficiently accurate are oesophagogastroduodenoscopy, including investigation for Helicobacter pylori, and abdominal ultrasonography. In cases where additional symptoms of IBS are present, endoscopic inspection of the colon is also recommended. These investigations are necessary when the medical history and symptoms are typical, and when preliminary laboratory tests such as blood count, electrolytes, hepatic and renal function, as well as erythrocyte sedimentation rate or CRP, and if applicable, peripheral thyroid parameters are within the normal range.

IRRITABLE BOWEL SYNDROME (IBS)

Introduction

Irritable Bowel Syndrome (IBS) is a functional gastrointestinal disorder characterized by altered bowel habits and abdominal pain in the absence of detectable structural abnormalities. *Formerly known* as spastic colon, mucous colitis, irritable colon, and spastic colitis, IBS has a *prevalence ranging from* 4.2% to 7.5% in India, with a higher incidence among females than *males, according to* epidemiological studies conducted in 2017.

Pathophysiology

Irritable bowel syndrome (IBS) is a complex disorder with unclear etiology, though multiple *factors are believed to contribute* to its development. Genetic predisposition plays a crucial *role, as certain individuals may* be more susceptible to IBS. Additionally, intestinal infections *such as gastroenteritis can trigger* symptoms, and food intolerances particularly to specific *carbohydrates, gluten, or lactose* are relevant. Psychosocial factors, including stress, anxiety, *and depression, significantly impact* the brain-gut axis, which regulates gastrointestinal *function. Disruptions in this* communication can lead to abnormal gut motility and heightened *sensitivity to food and stress.*

Environmental influences, such as dietary changes and hormonal fluctuations during menstruation or menopause, may also play a part. Modern lifestyles characterized by stress and inadequate fiber intake can further exacerbate IBS symptoms. Ultimately, the interplay of these factors can lead to changes in gut motility and sensory perception, resulting in the characteristic symptoms of IBS, including abdominal pain, bloating, and altered bowel habits.

Clinical features:

The most common presentation is recurrent lower abdominal pain or discomfort which is cramping or colicky in nature and relieved by defecation. Other features include abdominal distension, flatulence, dyspepsia, heartburn, sense of incomplete evacuation and alternate episodes of constipation and diarrhoea.

Irritable bowel syndrome is classified into four subtypes based on the bowel consistency that helps in focusing on treatment on the predominant type

- **IBS with predominant constipation (IBS-C):** tend to pass infrequent pellet (small, dry and hard) stools associated with abdominal pain or rectal pain.
- **IBS with predominant diarrhoea (IBS-D):** frequent defecation but produce small volumes of loose and watery stools associated with mucus
- **IBS with mixed bowel habits (IBS-M) or alternating IBS (IBS-A):** Alternating bouts of constipation and diarrhoea is the prime feature
- **Unsubtyped IBS (IBS-U):** is a specific type of IBS in which the stool consistency does not meet the criteria for IBS-C, D, or M. There is abnormal stool frequency (< 3 times/week or > 3 times/day).

Unintended weight loss, rectal bleeding, anemia, vomiting, fever, a family history of colorectal cancer, inflammatory bowel disease (IBD), coeliac disease, and onset of symptoms after the age of 50 are considered red flag features of IBS

Diagnosis:

Diagnosis of IBS relies on clinical evaluation, stool examination, and sigmoidoscopy or colonoscopy. Clinical diagnosis is established using the Rome IV criteria, i.e. recurrent abdominal pain or discomfort on average of at least 1 day per week in the last 3 months, associated with two or more of the following: improvement with defecation or associated with a change in stool frequency or associated with a change in stool form (appearance) (these criteria should be fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis). Stool examination detects occult blood or infections such as amoebae and giardia. Sigmoidoscopy or colonoscopy is performed to exclude colon cancer and inflammatory bowel disease (IBD)

INFLAMMATORY BOWEL DISEASES (IBD)

Introduction

Inflammatory bowel disease (IBD) is a collective term encompassing chronic conditions characterized by inflammation in the gastrointestinal (GI) tract. The two primary forms of

IBD are Crohn's disease and ulcerative colitis. These conditions present as recurring and alternating patterns of persistent inflammation in various areas of the gastrointestinal tract, leading to symptoms such as diarrhea and abdominal pain.

Crohn's Disease (CD)

Crohn's disease is a chronic transmural inflammatory bowel disease that usually affects the distal ileum and colon but may occur in any part of the gastrointestinal tract. Crohn's disease is also known as Regional Enteritis, Granulomatous Ileitis and Granulomatous Ileocolitis. Crohn's disease is more prevalent in Western nations. In contrast, ulcerative colitis (UC) is the more prevalent condition over Crohn's disease (CD) in developing countries such as India, with a UC/CD ratio of approximately 8:1.

Pathophysiology

Crohn's disease is a complex condition with multiple contributing factors, and despite ongoing research, its exact cause remains unknown. Current understanding suggests that it arises from an abnormal immune response to various environmental triggers in genetically predisposed individuals, such as certain drugs, toxins, infections, or intestinal microorganisms. This inappropriate immune response leads to chronic inflammation and damage in the gastrointestinal tract, resulting in the characteristic symptoms of Crohn's disease. The interplay between genetic susceptibility, immune system dysfunction, and environmental factors is believed to contribute to the development and progression of the disease.

Clinical features:

The clinical features of Crohn's disease commonly present as diarrhoea, abdominal pain, nausea, and vomiting. Systemic manifestations of the condition include weight loss, fever, and fatigue. Additionally, extra intestinal manifestations may involve the eyes (episcleritis, scleritis, uveitis), mouth (stomatitis, aphthous ulcers), liver (gallstones, cholangitis, primary sclerosing cholangitis), kidneys (nephrolithiasis, hydronephrosis, urinary tract infections), joints (axial (ankylosing spondylitis) or peripheral (knees, ankles, wrists, elbows) arthritis), and skin (erythema nodosum and pyoderma gangrenosum).

Complications:

Potential complications may include bowel obstruction, perforation, ulcers in the intestine, mouth and anus, perianal fistulas and abscesses, colorectal carcinoma, and toxic colitis.

Diagnosis:

Diagnosis of Crohn's disease is based on various assessments, including clinical evaluation, stool examination; abdominal CT, magnetic resonance enterography (MRE), upper endoscopy, colonoscopy, and video capsule endoscopy. The clinical evaluation may reveal signs of malnutrition, such as pallor and low weight, while vital signs assessment may uncover fever, tachycardia, and hypotension. Detailed abdominal inspection may reveal enterocutaneous fistulae in patients with advanced disease, with hyperactive bowel sounds suggesting inflammation or obstruction, and absent bowel sounds indicating ileus or severe inflammation. Palpation may demonstrate tenderness localized to the relevant intestinal area, and guarding may suggest peritonitis in individuals with intra-abdominal perforation or abscess. Furthermore, advanced perianal disease may manifest as perianal fistulae or hemorrhoids, necessitating a digital rectal examination.

Stool tests, including culture and sensitivities, ovum and parasites, and C. difficile toxins, are conducted to exclude infections, with stool calprotectin utilized to identify active Crohn's disease and monitor disease activity.

Imaging studies such as abdominal and pelvic computed tomography (CT) and magnetic resonance enterography (MRE) can detect abscesses, strictures, and fistulization. Visualization of the bowel mucosa is achievable through upper endoscopy and colonoscopy, enabling assessment of the extent of bowel inflammation and the collection of tissue samples for disease confirmation. In cases where regular endoscopic procedures cannot access the small bowel in patients with Crohn's disease, video capsule endoscopy (VCE) offers a valuable alternative for visualization

Ulcerative Colitis (UC)

Ulcerative colitis is the idiopathic, chronic and recurrent inflammatory disease affecting the mucosa and submucosa of the colon and rectum. It is a type of inflammatory bowel disease (IBD). Ulcerative colitis is also known as Colitis gravis. Epidemiological studies conducted in 2016 reported that the incidence of Ulcerative Colitis (UC) is increasing in India and in 2017 Indian Society of Gastroenterology reported that India has a higher incidence rate among Asian countries. Ulcerative colitis is commonly seen in both genders equally and prevalence is higher in the age group 20-39 years and above 60 years of age.

Pathophysiology

The exact cause of ulcerative colitis remains obscure but is believed to arise from multiple interrelated factors. Genetic predisposition significantly influences the immune system's function, increasing susceptibility. Psychological factors, such as stress, also contribute to the onset and exacerbation of ulcerative colitis. Environmental influences, including dietary habits and tobacco use, have been linked to ulcerative colitis development. Diets high in processed foods, sugars, and saturated fats may trigger inflammation while smoking elevates the risk of developing the disease. Infections affecting the gut microbiome can also provoke abnormal immune responses in susceptible individuals.

In ulcerative colitis, the immune system mistakenly targets the gut lining, leading to chronic inflammation in the mucosa and submucosa layers of the colon. This immune dysregulation results in an imbalance of pro-inflammatory and anti-inflammatory responses. Immune cells, including T cells and macrophages, produce cytokines that damage the gut lining, causing ulceration, bleeding, and diarrhoea. The chronic inflammation is characterized by immune cell activation and cytokine release, resulting in symptoms such as abdominal pain, diarrhoea, weight loss, and rectal bleeding.

Clinical features:

The commonest symptom of ulcerative colitis is loose stools containing blood and mucus. The presentation varies, depending on the site and severity of the disease.

Ulcerative colitis is classified according to site of the disease.

- **Ulcerative proctitis:** inflammation is confined to the rectum and it causes rectal bleeding and mucus discharge accompanied by tenesmus
- **Proctosigmoiditis:** inflammation involves the rectum and sigmoid colon and it causes bloody diarrhoea with mucus, often with abdominal cramps, pain in the left side of the abdomen and tenesmus.
- **Left sided colitis:** inflammation may reach up to the splenic flexure and it causes bloody diarrhoea with mucus, abdominal cramps and severe pain in the left side of the abdomen, loss of appetite and weight loss.
- **Pancolitis:** inflammation involves entire large intestine and it causes severe bloody diarrhea, abdominal cramps and pain, fatigue and significant weight loss. In severe cases, anorexia, malaise, anaemia, weight loss, fever, tachycardia may occur.

According to **Truelove-Witts criteria**, disease severity is categorized as

- **Mild:** bowel frequency is <4 /day with intermittent rectal bleeding
- **Moderate:** bowel frequency is 4-6/day with usual rectal bleeding may be associated with fever and abdominal pain.
- **Severe:** bowel frequency is >6/day with rectal bleeding associated with fever and abdominal pain.

Complications:

Complications associated with the ulcerative colitis include intestinal perforation, massive haemorrhage, toxic megacolon, fulminant colitis, stricture and colorectal carcinoma. Additionally, potential complications may involve primary sclerosing cholangitis, electrolyte imbalances, venous thrombosis, osteoporosis, arthritis in large joints, erythema nodosum, pyoderma gangrenosum, conjunctivitis, iritis, and episcleritis.

Diagnosis:

Diagnosis is based on the clinical evaluation, stool examination, barium enema, colonoscopy and flexible sigmoidoscopy with tissue biopsy. Clinical evaluation is contingent upon the patient's medical history, the presence of typical symptoms and signs, as well as potential extraintestinal manifestations or a history of prior similar episodes. Stool examination reveals the presence of blood and mucus within fecal matter, while microscopic examination demonstrates the presence of erythrocytes, neutrophils, and macrophages. Chronic ulcerative colitis can be identified through a double contrast barium enema X-ray, which illustrates the characteristic "stove-pipe" sign. Moreover, both colonoscopy and flexible sigmoidoscopy may reveal active inflammation along with the presence of pseudopolyps or a complicating carcinoma. To confirm the diagnosis, biopsies should be procured from the affected colon.

MCQ's

1. What is dyspepsia also known as

a) Heartburn b) Indigestion

c) Acid reflux d) Ulcer

2. What causes ulcer dyspepsia

a) Viral infections b) Peptic ulcer disease (PUD)

c) Food allergies d) Bacterial overgrowth

3. Which criteria are used to diagnose functional dyspepsia

a) Rome III criteria b) Rome IV criteria

c) Rome V criteria d) Rome VI criteria

4. Which subgroup of functional dyspepsia is characterized by predominant epigastric pain or burning

a) Postprandial Distress Syndrome (PDS) b) Irritable Bowel Syndrome (IBS)

c) Epigastric Pain Syndrome (EPS) d) Gastroesophageal Reflux Disease (GERD)

5. What are common clinical features of dyspepsia

a) Chronic cough and wheezing

b) Joint pain and swelling

c) Epigastric burning pain, abdominal discomfort, and nausea

d) Skin rash and itching

6. What characterizes Irritable Bowel Syndrome (IBS)

a) Detectable structural abnormalities in the GI tract

b) Altered bowel habits and abdominal pain without detectable structural abnormalities

c) Chronic liver disease

d) Persistent rash and itching

7. Which subtype of IBS is characterized by infrequent pellet stools and abdominal pain
   - a) IBS with predominant diarrhoea (IBS-D)
   - b) IBS with mixed bowel habits (IBS-M)
   - c) Unsubtyped IBS (IBS-U)
   - d) IBS with predominant constipation (IBS-C)
8. Which of the following are considered red flag features of IBS
   - a) Unintended weight loss, rectal bleeding, and anaemia
   - b) Chronic cough and wheezing
   - c) Joint pain and swelling
   - d) Persistent rash and itching
9. What common clinical features are associated with IBS
   - a) Chronic cough and wheezing
   - b) Joint pain and swelling
   - c) Recurrent lower abdominal pain, abdominal distension, and flatulence
   - d) Persistent rash and itching
10. What is Crohn's Disease also known as
    - a) Regional Enteritis
    - b) Granulomatous Ileitis
    - c) Granulomatous Ileocolitis
    - d) All of the above
11. What are common clinical features of Crohn's Disease
    - a) Chronic cough and wheezing
    - b) Joint pain and swelling
    - c) Diarrhea, abdominal pain, nausea, and vomiting
    - d) Persistent rash and itching
12. Which extra-intestinal manifestation is associated with Crohn's Disease
    - a) Chronic liver disease
    - b) Episcleritis, scleritis, and uveitis
    - c) Asthma and bronchitis
    - d) Chronic kidney disease

13. What are potential complications of Crohn's Disease
    - a) Chronic liver disease and asthma
    - b) Bowel obstruction, perforation, and colorectal carcinoma
    - c) Chronic kidney disease and diabetes
    - d) Persistent rash and itching
14. What is another name for Ulcerative Colitis
    - a) Regional Enteritis
    - b) Colitis gravis
    - c) Irritable Bowel Syndrome
    - d) Crohn's Disease
15. What is the most common symptom of Ulcerative Colitis
    - a) Chronic cough
    - b) Loose stools containing blood and mucus
    - c) Severe joint pain
    - d) Persistent rash
16. Which type of Ulcerative Colitis causes inflammation confined to the rectum
    - a) Proctosigmoiditis
    - b) Pancolitis
    - c) Ulcerative proctitis
    - d) Left sided colitis

Answer Keys

1	b	2	b	3	b	4	c	5	c	6	b	7	d	8	a
9	c	10	d	11	c	12	b	13	b	14	b	15	b	16	c

Short Answer Questions

1. Explain Dyspepsia
2. Explain Irritable bowel syndrome
3. Explain Crohn's disease
4. Explain Ulcerative colitis

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TOPIC 9

Mūtrakṛcchra

Introduction

Mūtrakṛcchra is Vāta prādhana Tridoṣaja vyādhi characterized by Duḥkhena mūtra pravṛttiḥ (difficulty or painful micturition). Mūtrakṛcchra occurs as an independent disease and also as a premonitory symptom and symptoms of other diseases. Ācārya Caraka described Mūtrakṛcchra in Trimarmīya cikitsitā as it is the disease of one among the three Marma (vital organs) i.e. Basti (urinary bladder) and also he described that Mūtrakṛcchra is one among the types of Mūtrāghāta. Ācārya Suśruta described Mūtrakṛcchra after Mūtrāghata because of their similarity in the Adhiṣṭhāna (seat i.e. Bastigata). Ācārya Vāgbhaṭa described Mūtrakṛcchra along with Mūtrāghata because both of these diseases arise from the Basti. In Rājanighaṇṭu, the synonyms of Mūtrakṛcchra are mentioned as Kṛcchraṃ, Mūtrarodha and Aśmarī. Ācārya Caraka and Ācarya Suśruta used Mūtrāghāta and Mūtropaghāta as a synonym of Mūtrakṛcchra respectively.

- मूत्रकृच्छ्रे कृच्छ्रत्वमधिकं विबन्धश्चाल्पः, मूत्राघाते तु विबन्धो बलवान् कृच्छ्रत्वमल्पश्चेति भेदः॥ (Āḍ.Śā.P.7/57)

Even though Ācāryas used Mūtrakṛcchra and Mūtrāghāta as synonymous, there is slight difference in them, i.e. In Mūtrakṛcchra; there is Kṛcchratvamadhika (severe discomfort or pain) and Vibandhaścālpa (less obstruction) whereas in Mūtrāghāta, there is Vibandho balavān (severe obstruction) and Kṛcchratvamalpa (less discomfort or pain)

Nirūkti and Paribhāṣā

The term Mūtrakṛcchra is composed of two words Mūtra and Kṛcchra. The word Mūtra means Prasrāva i.e. urine and Kṛcchra means Kaṣṭam i.e. difficult or trouble. Hence Mūtrakṛcchra means difficulty or troublesome micturition.

- मूत्रकृच्छ्रम् । मूत्रजन्यं कृच्छ्रमिति वा। (SKD)

That which causes Kṛcchraṃ (difficulty or troublesome) in Mūtrajanya (relating to urine) is called Mūtrakṛcchra

- मूत्रकृच्छ्रं दुःखेन मूत्रप्रवृत्तिः। (Ḍa.Su.U.59/1)
- यन्मूत्रकृच्छ्रे मूत्रं कृच्छ्रेण वहति । (Ca.Ca.Ci.26/43-44)

That which causes Duḥkhena mūtrapravṛtti or Mūtra kṛcchreṇa vahati (difficulty or painful micturition) is called Mūtrakṛcchra.

- मूत्रकृच्छ्राणीति मूत्रस्य कृच्छ्रेण महता दुःखेन प्रवृत्तिः। (Ma.M.N.30/1)
- मूत्रस्य कृच्छ्रेण महता दुःखेन प्रवृत्तिर्मूत्रकृच्छ्रम्। (Āḍ.Śā.P.7/57)

That which causes Mūtrasya kṛcchrena mahatā Duḥkhena pravṛtti (difficulty in micturition with severe discomfort) is called Mūtrakṛcchra

- नालस्य मूले मध्ये वा रुद्धं वाग्रे सदाहरुव्। कणशश्चेत्स्रावेन्मूत्रं मूत्रकृच्छ्रं तदुच्यते॥ (A.Ni.136)

That which causes Dāha (burning sensation) and Ruk (pain) in the Nālasya mūla madhye (base or middle or edge of the urethra) due to Ruddhaṃ (obstruction) associated with Kaṇaśaścetsravenmūtraṃ (urine is excreted drop by drop) is called Mūtrakṛcchra.

Sāmānya Nidāna and Saṃprāpti of Mūtrakṛcchra

- व्यायामतीक्ष्णौषधरूक्षमद्यप्रसङ्गनित्यद्रुतपृष्ठयानात्। आनूपमत्स्याध्यशनादजीर्णात् स्युर्मूत्रकृच्छ्राणि नृणामिहाष्टौ॥ (Ca.Ci.26/32)

Due to indulging in Vyāyāma (exercise), Tīkṣṇāuṣadha (sharp medicines), Rūkṣa (non-unctuous substances), Madya (intake of alcohol), Prasaṅganityadrutapṛṣṭhayānāt (riding on the back of fast moving animals), Ānūpa (meat of marshy land animals), Matsya (fish), Adhyāśna (eating before digestion of previous food) and Ajīrṇāt (indigestion), manifestation of eight types of Mūtrakṛcchra occurs.

- पृथङ्मलाः स्वैः कुपिता निदानैः सर्वेऽथवा कोपमुपेत्य बस्तौ। मूत्रस्य मार्गं परिपीडयन्ति यदा तदा मूत्रयतीह कृच्छ्रात्॥ (Ca.Ci.26/33)

Due to aforesaid causative factors, the doṣas gets aggravated due to their own respective causative factors, or else all the three doṣas gets aggravated together in the Basti (urinary bladder) and does Mūtra mārgaṃ paripīdana (injuring or damaging the urinary passage) resulting in Mūtrakṛcchra.

Saṃprāpti Ghaṭaka of Mūtrakṛcchra

Doṣa	Vāta Pradhāna Tridoṣa
Dūṣya	Rasa, Rakta, Śukra, Purīṣa and Mūtra
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma

Srotas	Mūtra vaha
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Mūtra mārga
Udbhava sthāna	Āmapakvāśaya
Vyakta sthāna	Basti
Adhiṣṭhāna	Basti
Rogamārga	Madhyama

Bheda of Mūtrakṛcchra

Ācārya Vāgbhaṭa classified Mūtrakṛcchra in to four types namely Vātaja, Pittaja, Kaphaja, and Sannipātaja based on the predominance of doṣas.

- अष्टौ मूत्राघाता इति वातपित्तकफसन्निपाताश्मरीशर्कराशुक्रशोणितजाः (Ca.Sū.19/4(1))

Ācārya Caraka classified Mūtrakṛcchra in to eight types namely Vātaja, Pittaja, Kaphaja, Sannipātaja, Aśmarija, Śarkarāja, Śukraja and Śoṇitaja or Kṣataja Mūtrakṛcchra.

- वातेन पित्तेन कफेन सर्वैस्तथाऽभिघातैः शकृदश्मरीभ्याम् । तथाऽपरः शर्करया सुकष्टो मूत्रोपघातः कथितोऽष्टमस्तु ॥ (Su.U.59/3)

Even Ācārya Suśruta classified Mūtrakṛcchra in to eight types namely Vātaja, Pittaja, Kaphaja, Sarvaja, Abhighātaja, Śakṛdaja, Aśmarīja and Śarkarāja Mūtrakṛcchra

Mūtrakṛcchra Bheda	Caraka	Suśruta	Vāgbhaṭa	Mādhava
Vātaja	+	+	+	+
Pittaja	+	+	+	+
Kaphaja	+	+	+	+
Sannipātaja	+	+	+	+
Aśmarīja	+	+	–	+
Śarkarāja	+	+	–	–
Śukraja	+	–	–	+
Śakṛdaja	–	+	–	+
Śoṇitaja	+	–	–	–
Abhighātaja	–	+	–	–
Śalyaja	–	–	–	+

Bheda Lakṣaṇās

Vātaja Mūtrakṛcchra

- तीव्रा रुजो वङ्क्षणबस्तिमेढ्रे स्वल्पं मुहुर्मूत्रयतीह वातात्। (Ca.Ci.26/34)
- बस्तिवङ्क्षणमेढ्रार्तियुक्तोऽल्पाल्पं मुहुर्मुहुः। मूत्रयेद्वातजे कृच्छ्रे... । (A.H.Ni.9/4)

The clinical features of Vātaja Mūtrakṛcchra are Tīvra ruja (severe pain) in Vaṅkṣaṇa (groin), Basti (bladder) and Meḍhra (penis), and person passes Svalpa muhurmūtra (frequently little quantity of urine)

- अल्पमल्पं समुत्पीड्य मुष्कमेहनबस्तिभिः । फलद्भिरिव कृच्छ्रेण वाताघातेन मेहति ॥ (Su.U.59/4)

Ācārya Suśruta mentioned Alpamalpa (passes urine in little quantity) and Samutpīḍya muṣkamehanabastibhiḥ (tearing pain in the scrotum, penis and the bladder) are the clinical features of Vātaja Mūtrakṛcchra.

Pittaja Mūtrakṛcchra

- पीतं सरक्तं सरुजं सदाहं कृच्छ्रान्मुहुर्मूत्रयतीह पित्तात्॥ (Ca.Ci.26/34)
- ...पैत्ते पीतं सदाहरुक्॥ रक्तं वा ... । (A.H.Ni.9/4-5)

The clinical features of Pittaja Mūtrakṛcchra are Pītaṃ (yellowish) or Saraktaṃ (reddish urine) along with Sarujaṃ (pain), Sadāhaṃ (burning sensation) and Kṛcchrān muhurmūtra (passes urine frequently with difficulty).

- हारिद्रमुष्णं रक्तं वा मुष्कमेहनबस्तिभिः । अग्निना दह्यमानाभैः पित्तघातेन मेहति ॥ (Su.U.59/5)

Ācārya Suśruta mentioned Hāridramuṣṇaṃ raktaṃ vā (passes yellowish or reddish urine with hot sensation) and Muṣkamehanabastibhiḥ Agninā dahyamānābhaiḥ (feels burning sensation in the scrotum, penis and the bladder as if burnt with fire) are the clinical features of Pittaja Mūtrakṛcchra.

Kaphaja Mūtrakṛcchra

- बस्तेः सलिङ्गस्य गुरुत्वशोथौ मूत्रं सपिच्छं कफमूत्रकृच्छ्रे। (Ca.Ci.26/35)
- ...कफजे बस्तिमेढ्रगौरवशोफवान्। सपिच्छं सविबन्धं च ...। (A.H.Ni.9/5)

The clinical features of Kaphaja Mūtrakṛcchra are Bastiḥ saliṅgasya gurutvaśothau (heaviness and swelling in the bladder and the penis) and Mūtrasapicchaṃ (passes slimy urine).

- स्निग्धं शुक्लमनुष्णं च मुष्कमेहनबस्तिभिः । संहृष्टरोमा गुरुभिः श्लेष्माघातेन मेहति ॥ (Su.U.59/6)

Ācārya Suśruta mentioned Snigdhaṃ śuklamanuṣṇaṃ (passes glossy, white urine which is not warm), Saṃhṛṣṭaromā (horripilation) and Muṣkamehanabastibhiḥgurubhiḥ (feels heaviness in the scrotum, penis and the bladder) are the clinical features of Kaphaja Mūtrakṛcchra

Sannipātaja Mūtrakṛcchra

- सर्वाणि रूपाणि तु सन्निपाताद्भवन्ति तत् कृच्छ्रतमं हि कृच्छ्रम्॥ (Ca.Ci.26/35)
- ....सर्वैः सर्वात्मकं मलैः॥ (A.H.Ni.9/5)

The clinical features of Sannipātaja Mūtrakṛcchra are Sarvāṇi rūpaṇi (all the features of three doṣas manifested together) and it is considered as Kṛcchratamaṃ (most difficult to cure).

- दाहशीतरुजाविष्टो नानावर्णं मुहुर्मुहुः । ताम्यमानस्तु कृच्छ्रेण सान्निपातेन मेहति ॥ (Su.U.59/7)

Ācārya Suśruta mentioned Dāhaviṣṭa (burning sensation in the urethra etc.), Śītāviṣṭa (cold or shivering of the body), Rujāviṣṭa (painful urination), Nānavarṇa muhurmuhuḥ kṛcchreṇa (frequently passes urine in varied colors and with difficulty) and Tāmyamānastu (feels darkness before the eyes) are the clinical features of Sannipātaja Mūtrakṛcchra

Aśmarīja and Śarkarāja Mūtrakṛcchra

- विशोषयेद्बस्तिगतं सशुक्रं मूत्रं सपित्तं पवनः कफं वा । यदा तदाऽश्मर्युपजायते तु क्रमेण पित्तेष्विव रोचना गोः॥ (Ca.Ci.26/36)

In case of Aśmarīja Mūtrakṛcchra, the Vāta localized in the Basti (bladder) dries up the Śukra, Mūtra along with Pitta or Kapha and develops the Aśmari (stone or calculi) gradually like that of formation of Gorocana (gall stone in the cows) due to gradual desiccation of Pitta (bile).

- कदम्बपुष्पाकृतिरश्मतुल्या श्लक्ष्णा त्रिपुट्यप्यथवाऽपि मृद्वी। मूत्रस्य चेन्मार्गमुपैति रुद्ध्वा मूत्रं रुजं तस्य करोति बस्तौ॥ ससेवनीमेहनबस्तिशूलं विशीर्णधारं च करोति मूत्रम्। मृद्नाति मेढ्रं स तु वेदनार्तो मुहुः शकृन्मुञ्चति मेहते च॥ क्षोभात् क्षते मूत्रयतीह सासृक् तस्याः सुखं मेहति च व्यपायात्। (Ca.Ci.26/37-39)

Calculus which resembles the shape of Kadambapuṣpākṛti (the flower of Kadamba), Aśmatulyā (stony hard), Ślakṣṇa (smooth), Triputī (three layered) or Mṛdvī (soft) reaches the Mūtramārga (urinary passage) and does Ruddhvā mūtra (obstruction to the flow of urine) and produces Ruja (pain) in the Basti (urinary bladder). And also it produces Śūla (pain) in the Sasevaṇī (perineal region), Mehana (penis) and Basti (supra pubic region), Viśīrṇadhāraṃ mūtra (divided stream of urine), Mṛdnāti meḍhra vedanārte (due to pain, the patient rubs or squeezes the penis) and Muhuḥ śakṛnmuñcati mehate (passes faeces and urine frequently). Kṣobhāt kṣate (due to irritation caused by calculus), the patient develops Kṣata (injury in the urinary passage or bladder) and passes Sāsṛk mūtra (urine mixed with blood). When calculus gets Vyapāyāt (expelled from urinary passage), the person passes Sukhaṃ mehati (urine comfortably).

- एषाऽश्मरी मारुतभिन्नमूर्तिः स्याच्छर्करा मूत्रपथात् क्षरन्ती॥ (Ca.Ci.26/39)

When the Aśmarī (calculus) become Bhinnamūrti (broken in to small particles) due to aggravated Vāta, then such particles are called as Śarkarā, which is Mūtrapathāt kṣaranti (discharges with urine).

- पच्यमानस्य पित्तेन भिद्यमानस्य वायुना । श्लेषमणोऽवयवा भिन्नाः शर्करा इति सञ्ज्ञिताः ॥
  हृत्पीडा वेपथुः शूलं कुक्षौ वह्निः सुदुर्बलः । ताभिर्भवति मूर्च्छा च मूत्राघातश्च दारुणः ॥
  मूत्रवेगनिरस्तासु तासु शाम्यति वेदना । यावदन्या पुनर्नैति गुडिका स्रोतसो मुखम् ॥
  शर्करासम्भवस्यैतन्मूत्राघातस्य लक्षणम् । (Su.U.59/12-15)

When the Aśmarī gets transformed by Pitta (Pacyamānasya Pittena), then splitted by Vāta (Bhidyamānasya Vāta) and disintegrated or loosened by Kapha (Sleṣmaṇoavayavā), they are called Śarkarā (gravels), which leads to Hṛtpīḍa (pain in the cardiac region), Vepathu (tremors), Kukṣi śūla (abdominal pain), Durbalavahni (impaired digestive fire), Mūrcchā (fainting) and Dāruṇa mūtrāghāta (severe painful micturition). When the Mūtraveganiras (gravels expelled out with urine), the pain subsides to the last till Punarnaiti guṭikā (another gravel) does not obstructs the Srotaso mukham (opening of the passage). These are the clinical features of Śarkarāja Mūtrakṛcchra.

Śukraja Mūtrakṛcchra

- शुक्रं मलाश्चैव पृथक् पृथग्वा मूत्राशयस्थाः प्रतिवारयन्ति। तद्व्याहतं मेहनबस्तिशूलं मूत्रं सशुक्रं कुरुते विबद्धम्॥ स्तब्धश्च शूनो भृशवेदनश्च तुद्येत बस्तिर्वृषणौ च तस्य। (Ca.Ci.26/42-43)

The aggravated Doṣas located in the Mūtrāśaya (urinary bladder) either single or all together does the Śukra prativārayanti (prevention of the semen expulsion). Due to this, there will be manifestation of Mehanabasti śula (pain in the penis and urinary bladder), Mūtra saśukraṃ vibaddham (obstruction to the flow of urine and semen) along with Stabdha (stiffness), Śūna (swelling), Bṛśavedana (excessive pain) and Tudyet (pricking pain) in the Bastirvṛṣaṇā (urinary bladder and scrotum).

Kṣataja or Raktaja Mūtrakṛcchra

- क्षताभिघातात् क्षतजं क्षयाद्वा प्रकोपितं बस्तिगतं विबद्धम्॥ तीव्रार्ति मूत्रेण सहाश्मरीत्वमायाति तस्मिन्नतिसञ्चिते च। आध्माततां विन्दति गौरवं च बस्तेर्लघुत्वं च विनिःसृतेऽस्मिन्॥ (Ca.Ci.26/43-44)

Due to Kṣatābhighāta (wound caused by injury) or Kṣaya (loss of semen), the Kṣataja (Rakta) gets aggravated in the Basti (bladder) and causes Vibaddha (obstruction), Tīvrārti (severe pain), Mūtreṇa sahāśmarītvamāyāti tasminnatisañcite (urine becomes similar to calculus and gets accumulated in the bladder) and produces Ādhmātatāṃ vindati gauravaṃ (distension and heaviness of abdomen) and feels Basterlaghutvaṃ (lightness of bladder) when the calculus gets Viniḥsṛte (departed out).

- मूत्रवाहिषु शल्येन क्षतेष्वभिहतेषु च । स्रोतःसु मूत्राघातस्तु जायते भृशवेदनः ॥
  वातबस्तेस्तु तुल्यानि तस्य लिङ्गानि लक्षयेत् । (Su.U.59/8-9)

Kṣata (injury) to the Mūtravaha srotas (urinary passage) by Śalya (foreign body) or Abhihateṣu (trauma) produces Srotaḥsu Mūtrāghāta (obstruction to the urinary passage) with Bhṛśavedana (severe pain). And also the clinical features which are similar to those of Vātabasti are manifested.

Śakṛdaja Mūtrakṛcchra

- शकृतस्तु प्रतीघाताद्वायुर्विगुणतां गतः ॥ आध्मानं च सशूलं च मूत्रसङ्गं करोति हि । (Su.U.59/9-10)

Due to Śakṛtastu pratighātāt (obstruction of faeces), the Vāta gets aggravated and moves reverse direction and produces Ādhmāna (distension of abdomen) with Saśūla (colic pain) and Mūtrasaṅga (retention of urine).

Sādhyāsādhyata of Mūtrakṛcchra

- द्विदोषोद्भवं मूत्रकृच्छ्रं सदाहं भवेत्कष्टसाध्यं प्रयत्नौषधीभिः।
  त्रिदोषोत्थितं दारुणं प्राणनाशं निरुत्तं मुनीन्द्रैरसाध्यं नितान्तम्॥ (H.Ni.34/6)

Mūtrakṛcchra caused by Dvidoṣaja (involvement of two doṣas) associated with Dāha (burning sensation) is Kaṣṭasādhya (difficult to cure) and should be treated with special Auṣadhi (medicines). Mūtrakṛcchra caused by Tridoṣa (involvement of three doṣas) and Dāruṇa (in severe form) is considered to be Prāṇanāśa (fatal) and the experts opine that it is Asādhya (incurable) even by extraordinary efforts.

In Contemporary science,

Mūtrakṛcchra can be correlated with dysuria (painful micturition), which is commonly seen as a symptom of infective conditions like urinary tract infection (urethritis, cystitis, prostatitis, ureteritis and pyelonephritis), Renal calculi, chronic renal disease, etc., in contemporary science. For understanding purpose, here Mūtrakṛcchra is correlated to urinary tract infection in modern science. (Please refer Topic 11)

MCQ's

1. Duḥkhena mūtra pravṛttiḥ is the feature of
   a) Mūtrakṛcchra b) Mūtrāghata
   c) Both a and b d) None of the above
2. According to Vāgbhaṭa, Mūtrakṛcchra is of .... types
   a) 4 b) 5
   c) 6 d) 8

3. According to Suśruta, Mūtrakṛcchra is of .... types
   a) 4 b) 5
   c) 6 d) 8
4. कदम्बपुष्पाकृतिरश्मतुल्या is the feature of .... Mūtrakṛcchra
   a) Aśmarīja b) Śukraja
   c) Śoṇitaja d) Śarkaraja
5. Mūtrasaṅga is the feature of .... Mūtrakṛcchra
   a) Aśmarīja b) Śukraja
   c) Śakṛdaja d) Śarkaraja

Answer Keys

1	a	2	a	3	d	4	a	5	c

Short Answer Questions

1. Define Mūtrakṛcchra and explain the Nidāna and Samprāpti of Mūtrakṛcchra
2. Explain the types of Mūtrakṛcchra
3. Explain Aśmarīja and Śarkarāja Mūtrakṛcchra

◆

TOPIC 10

Mūtrāghāta

Introduction

Mūtrāghāta is Vāta Pradhāna Tridoṣaja Vyādhi characterized by Mūtrāvarodhaḥ (obstruction to the flow of urine). As already mentioned, that there is Vibandho balavān (severe obstruction) and Kṛcchratvamalpa (less discomfort or pain) predominant in Mūtrāghāta. Ācārya Caraka described Mūtradoṣa in Trimarmīyāṃ Siddhiṃ which are similar to that of Mūtrāghata as described by Ācārya Suśruta in Uttaratantra. Ācārya Cakrapāṇi opines that some scholars include Mūtraghāta as a specific stage of Mūtrakṛcchra.

Ācārya Vāgbhaṭa described Mūtrāghāta after Atīsāragrahaṇi nidāna and also he has categorized the Mūtradoṣas in to two types namely Mūtra atipravṛttijā (excessive elimination of urine) which include Prameha; and Mūtrāpravṛttijā (non-elimination of urine) which includes Mūtrāghāta, Mūtrakṛcchra and Aśmarī.

Nirūkti and Paribhāṣā

- मूत्राघात – मूत्रस्याघातो निरोधो येन्। (SKD)

That which causes Mūtra nirodha (obstruction or retention of urine) is called Mūtrāghāta.

- मूत्राघातो मूत्रावरोधः (Ḍa.Su.U.58/1)

That which causes Mūtrāvarodha (obstruction or retention of urine) is called Mūtrāghāta

- मूत्राघाते मूत्रं शोष्यते प्रतिहन्यते वा । (Ca.Ca.Ci.26/43-44)

That which causes Mūtra śoṣyate (desiccation of urine) or Mūtra pratihanyate (obstruction or retention of urine) is called Mūtrāghāta.

- मूत्रस्याभिघातो मूत्राघातः। आघातो वेदनाविशेषवान् मूत्रनिरोध इत्यपरे, स च वातादिदोषजनितः॥ (Āḍ.Śa.P.7/53)

Abhighāta (injury or damage) means Āghāta and it is described as Vedanaviśeṣavān mūtranirodha (specific type of distress or obstruction of urine). Therefore, Mūtrāghāta is specific type of distress or obstruction of urine due to vitiation of Vātādi doṣas

Sāmānya Nidāna and Saṃprāpti of Mūtrāghāta

Ācārya Caraka, Suśruta and Vāgbhaṭa have not described separate causative factor for Mūtrāghāta. Hence the causative factors of Mūtravaha srotoduṣṭi and Mūtrakṛcchra may be taken as general causative factors of Mūtrāghāta.

- जायन्ते कुपितैर्दोषैमूत्राघातात्रयोदश। प्रायो मूत्रविघाताद्यैर्वातकुण्डलिकाऽऽदयः॥ (B.P.M.36/1)

The Vātādi doṣas gets aggravated and produces thirteen types of Mūtrāghāta. Prominently the diseases like Vātakuṇḍalikā etc., are manifested due to Mūtravighāta (suppression of the urine).

Saṃprāpti Ghaṭaka of Mūtrāghāta

Doṣa	Vāta Pradhāna Tridoṣa
Dūṣya	Rasa, Rakta, Kleda, Sveda and Mūtra
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Mūtra vaha
Srotoduṣṭi	Saṅga, Vimārga gamana and Sirāja granthi
Sañcāra sthāna	Mūtra mārga
Udbhava sthāna	Āmapakvāśaya
Vyakta sthāna	Meḍhra, especially during the act of micturition
Adhiṣṭhāna	Basti
Rogamārga	Madhyama

Bheda of Mūtrāghāta

- मूत्रौकसादो जठरं कृच्छ्रमुत्सङ्गसङ्क्षयौ। मूत्रातीतोऽनिलाष्ठीला वातबस्त्युष्णमारुतौ॥ वातकुण्डलिका ग्रन्थिर्विड्घातो बस्तिकुण्डलम्। त्रयोदशैते मूत्रस्य दोषास्ताँल्लिङ्गतः शृणु॥ (Ca.Si.9/25-26)

Ācārya Caraka classified Mūtradoṣa or Mūtrāghāta in to thirteen types namely

Mutraukasāda, Mūtrajaṭhara, Mūtrakṛcchra, Mutrotsaṅga, Mūtrasaṅkṣaya, Mūtrātītā, Anilāṣṭhīlā, Vātabasti, Uṣṇamāruta, Vātakuṇḍalikā, Granthi, Viḍghāta and Bastikuṇḍala.

- वातकुण्डलिकाऽष्ठीला वातबस्तिस्तथैव च । मूत्रातीतः सजठरो मूत्रोत्सङ्गः क्षयस्तथा ॥ मूत्रग्रन्थिर्मूत्रशुक्रमुष्णवातस्तथैव च । मूत्रौकसादौ द्वौ चापि रोगा द्वादश कीर्तिताः ॥ (Su.U.58/3-4)

Ācārya Suśruta classified Mūtrāghāta in to twelve types namely Vātakuṇḍalikā, Aṣṭhila, Vātabasti, Mūtrātītā, Mūtrajaṭhara, Mutrotsaṅga, Mūtrakṣaya, Mūtragranthi, Mūtraśukra, Uṣṇavāta, Pittaja Mutraukasāda and Kaphaja Mutraukasāda

Ācārya Vāgbhaṭa classified Mūtrāghāta in to thirteen types namely Vātabasti, Vātāṣṭhila, Vātakuṇḍalikā, Mūtrātīta, Mūtrajaṭhara, Mutrotsaṅga, Mūtragranthi, Mūtraśukra, Viḍvighāta, Uṣṇavāta, Mūtrakṣaya, Pittaja Mutraukasāda and Kaphaja Mutraukasāda. But Ācārya Haṃsarāja classified Mūtrāghāta in to four types Vātaja, Pittaja, Kaphaja and Dvidoṣaja or Tridoṣaja.

Mūtrāghāta	Caraka	Suśruta	Vāgbhaṭa
Mutraukasāda / Mūtrasāda	+	+ (Kapha and Pittaja)	+ (Kapha and Pittaja)
Mūtrajaṭhara	+	+	+
Mūtrakṛcchra	+	–	–
Mutrotsaṅga	+	+	+
Mūtrasaṅkṣaya / Mūtrakṣaya	+	+	+
Mūtrātītā	+	+	+
Anilāṣṭhīlā / Vātāṣṭhīlā	+	+	+
Vātabasti	+	+	+
Uṣṇamāruta / Uṣṇavāta	+	+	+
Vātakuṇḍalikā	+	+	+
Granthi / Mūtragranthi	+	+	+
Viḍvighāta	+	–	+
Bastikuṇḍala	+	–	–
Mūtraśukra	–	+	+

Mūtrāghāta in terms of Mūtra Śoṣaṇa (desiccation of urine) or Mūtra pratihanyate (obstruction or retention of urine):

Mūtrāghāta	Mūtra Śoṣaṇa	Mūtra Pratihanyate
Mutraukasāda / Mūtrasāda	+	–
Mūtrajaṭhara	+	+
Mūtrakṛcchra	+	–
Mutrotsaṅga	+	+
Mūtrasaṅkṣaya / Mūtrakṣaya	+	–
Mūtrātītā	+	+
Anilāṣṭhīlā / Vātāṣṭhīlā	+	+
Vātabasti	–	+
Uṣṇamāruta / Uṣṇavāta	+	–
Vātakuṇḍalikā	+	+
Granthi / Mūtragranthi	+	+
Viḍvighāta	+	–
Bastikuṇḍala	+	+
Mūtraśukra	+	–

Mūtrāghāta Bheda Lakṣaṇās

Mūtraukasāda

- पित्तं कफो द्वावपि वा बस्तौ संहन्यते यदा। मारुतेन तदा मूत्रं रक्तं पीतं घनं सृजेत्॥
  सदाहं श्वेतसान्द्रं वा सर्वैर्वा लक्षणैर्युतम्। मूत्रौकसादं तं विद्यात् पित्तश्लेष्महरैर्जयेत्॥ (Ca.Si.9/27-28)

Pitta and Kapha either individually or combined gets aggravated with Vāta in the Basti (bladder) and produces the disease Mūtraukasāda, in which the patient voids urine in Raktaṃ (reddish), Pītaṃ (yellowish), Ghana (thick) and associated with Dāha (burning sensation) or Śveta (whitish), Sāndraṃ (dense or thick) urine or with Sarva lakṣaṇa (all the symptoms). This condition should be treated with Pittaśleṣmahara (measures alleviating Pitta and Kapha).

- विशदं पीतकं मूत्रं सदाहं बहलं तथा । शुष्कं भवति यच्चापि रोचनाचूर्णसन्निभम् ॥
  मूत्रौकसादं तं विद्याद्रोगं पित्तकृतं बुधः । पिच्छिलं संहतं श्वेतं तथा कृच्छ्रप्रवर्तनम् ॥
  शुष्कं भवति यच्चापि शङ्खचूर्णप्रपाण्डुरम् । मूत्रौकसादं तं विद्यादामयं द्वादशं कफात् ॥ (Su.U.58/24-26)

Ācārya Suśruta classified Mūtraukasāda into two types based on the predominance of doṣas, i.e. Pittaja and Kaphaja.

- In **Pittaja Mūtraukasāda,** the patient voids Bahala (copious) urine which is Viśada (clear) and Pītaka (yellowish) in colour associated with Dāha (burning sensation) and the urine resembles Rocanācūrṇa sannibha (the powder of cow's gall stone) when it becomes Śuṣka (dried).
- In **Kaphaja Mūtraukasāda,** the patient voids Picchilaṃ (slimy) and Saṃhataṃ (Ghana- thick) urine which is Śveta varṇa (whitish in colour) and Kṛcchrapravartana (passed with difficulty) and the urine resembles Śaṅkhacūrṇaprapāṇḍuram (pale white like conch shell powder) when it becomes Śuṣka (dried).

Mūtrajaṭhara

- ⊙ विधारणात् प्रतिहतं वातोदावर्तितं यदा। पूरयत्युदरं मूत्रं तदा तदनिमित्तरुक्॥
  अपक्तिमूत्रविट्सङ्गैस्तन्मूत्रजठरं वदेत्। (Ca.Si.9/29-30)

Due to Vidhāraṇāt (suppression of manifested urge of urine), the Vāta gets Pratihata (obstructed) and causes Udāvarta (upward movements of Vāta) resulting in Pūrayatyudaraṃ mūtraṃ (filling of abdomen with urine) and produces Animittaruk (unconditioned or severe pain), Apakti (indigestion) and Mūtraviṭsaṅga (obstruction of urine and faeces). This condition is known as Mūtrajaṭhara.

- ⊙ मूत्रस्य विहते वेगे तदुदावर्तहेतुना । अपानः कुपितो वायुरुदरं पूरयेद्भृशम् ॥
  नाभेरधस्तादाध्मानं जनयेत्तीव्रवेदनम् । तं मूत्रजठरं विद्यादधःस्रोतोनिरोधनम् ॥ (Su.U.58/13-14)

Mūtravega vihata (suppressing the urge of micturition) causes Udāvarta (upward movements of Vāta) resulting in aggravation of Apāna vāta and it leads to Udaraṃ pūrayedbhṛśa (severe distention of abdomen). This Nābheradhastādādhmāna (distension of abdomen below umbilical region) produces Tīvravedana (severe pain). This condition is known as Mūtrajaṭhara and it is due to Adhaḥ srotonirodhanam (obstruction of the lower channels).

Mūtrakṛcchra

- ⊙ मूत्रितस्य व्यवायात्तु रेतो वातोद्धतं च्युतम्। पूर्वं मूत्रस्य पश्चाद्वा स्रवेत् कृच्छ्रं तदुच्यते॥ (Ca.Si.9/32)

Due to Mūtritasya vyavāyāttu (indulgence in sexual intercourse during the urge of micturition), the Retas (semen) gets Cyuta (Svasthānāccyuta - dislodged from its site) by Vātoddhataṃ (agitated Vāta) and gets ejaculated Pūrva (before) or Paścāt (after) the passage of urine. This condition is known as Mūtrakṛcchra.

Mutrotsaṅga

- ⊙ खवैगुण्यानिलाक्षेपैः किञ्चिन्मूत्रं च तिष्ठति। मणिसन्धौ स्रवेत् पश्चात्तदरुग्वाऽथ चातिरुक्॥
  मूत्रोत्सङ्गः स विच्छिन्नमुच्छेषगुरुशेफसः । (Ca.Si.9/33-34)

Due to Khavaiguṇya (deformity in the urethra) and Anilākṣepa (aggravation of Vāta), the Kiñcinmūtra (little urine) gets Tiṣṭati (stagnated) in the Maṇisandhi (splincter of penis) and it Sravet paścāttadarugvāatha cātiruk (discharges later without or with severe pain). The Uccheṣamūtra (stagnated or residual urine) produces Vicchinna mūtra (urine flows with interruptions) from the Guruśepha (heavy phallus). This condition is known as Mutrotsaṅga.

- ⊙ बस्तौ वाऽप्यथवा नाले मणौ वा यस्य देहिनः । मूत्रं प्रवृत्तं सज्जेत सरक्तं वा प्रवाहतः ॥
  स्रवेच्छनैरल्पमल्पं सरुजं वाऽथ नीरुजम् । विगुणानिलजो व्याधिः स मूत्रोत्सङ्गसञ्ज्ञितः ॥ (Su.U.58/15-16)

When the flow of urine gets obstructed at the level of Basti (bladder) or Nāla (Meḍhra srotas - urethra) or Maṇi (Meḍhrāgradeśa - glans penis) and if the person strains to pass the urine, he passes Alpamalpa (little by little) associated with Rakta (blood) with pain or without pain due to Anilaviguṇa (vitiation of Vāta). This disease is called as Mutrotsaṅga.

Mūtrasaṅkṣaya

- ⊙ वाताकृतिर्भवेद्वातान्मूत्रे शुष्यति सङ्क्षयः॥ (Ca.Si.9/34)

Due to aggravation of Vāta, the Mūtra (urine) gets Śuṣyati (dries up) and produces the Vātākṛti (features of aggravated Vāta) and this condition is known as Mūtrasaṅkṣaya.

- ⊙ रूक्षस्य क्लान्तदेहस्य बस्तिस्थौ पित्तमारुतौ । सदाहवेदनं कृच्छ्रं कुर्यातां मूत्रसङ्क्षयम् ॥ (Su.U.58/17)

The aggravated Pitta and Vāta in the Basti (bladder) of the Rūkṣasya klānta deha (rough and exhausted or fatigued person) produces the disease Mūtrasaṅkṣaya (i.e. Mūtraśoṣaṇa (drying up of the urine)) which causes Dāha (burning sensation), Vedana (pain) and Mūtrakṛcchra (painful micturition).

Mūtrātītā

- ⊙ चिरं धारयतो मूत्रं त्वरया न प्रवर्तते। मेहमानस्य मन्दं वा मूत्रातीतः स उच्यते॥ (Ca.Si.9/35)

Due to Ciraṃ dhārayato mūtraṇ (suppressing the urgency of micturition for long time) there will be Tvarayā na pravartate (incapability to pass urine immediately) or Mehamānasya mandaṃ (the urine flows slowly). This condition is known as Mūtrātītā.

- ⊙ वेगं सन्धार्य मूत्रस्य यो भूयः स्रष्टुमिच्छति । तस्य नाभ्येति यदि वा कथञ्चित्सम्प्रवर्तते ॥
  प्रवाहतो मन्दरुजमल्पमल्पं पुनः पुनः । मूत्रातीतं तु तं विद्यान्मूत्रवेगविघातजम् ॥ (Su.U.58/11-12)

When a person has Vegaṃ sandhārya mūtrasya (suppressed the urgency of micturition) and again if he desires to pass the urine (Sraṣṭumicchati), the urine does not flow (Nābhyeti - Nāgacchatī) or if he strains to pass the urine, it flows Alpamalpaṃ (little by little) and Punaḥpunaḥ (again and again) with Mandaruja (mild pain). This condition is known as Mūtrātītā which is caused by Mūtravegavighātaja (suppression of the urge of micturition)

Anilāṣṭhīlā or Vātāṣṭhīlā

- ⊙ आध्मापयन् बस्तिगुदं रुद्ध्वा वायुश्चलोन्नताम्। कुर्यात्तीव्रार्तिमष्ठीलां मूत्रविण्मार्गरोधिनीम्॥ (Ca.Si.9/36)

The aggravated Vāta gets Bastiguda ruddhvā (obstructed in the bladder and rectum) causes Ādhmāpa (distension of bladder) and produces the disease Aṣṭhīlā (stone like hard mass) which is Cala (mobile), Unnatā (elevated) and causes Tīvrārti (severe pain) and Mūtraviṇmārgarodhinīm (obstruction of the urine and faecal passages).

- ⊙ शकृन्मार्गस्य बस्तेश्च वायुरन्तरमाश्रितः । अष्ठीलावद्घनं ग्रन्थिं करोत्यचलमुन्नतम् ॥
  विण्मूत्रानिलसङ्गश्च तत्राध्मानं च जायते । वेदना च परा बस्तौ वाताष्ठीलेति तां विदुः । (Su.U.58/7-8)

The aggravated Vāta gets localized in between the Śakṛnmārga (rectum) and Basti (bladder) produces Ghana (thick), Acala (Īṣaccala – slightly mobile), Unnatam (elevated) and Aṣṭilāvat granthi (stone like swelling) resulting in Viṇmūtrānilasaṅga (obstruction of faeces, urine and flatus), Ādhmāna (distension of the bladder) and Basti vedanā (pain in the bladder). This condition is known as Vāṭāṣṭhilā.

Vātabasti

⊙ मूत्रं धारयतो बस्तौ वायुः क्रुद्धो विधारणात्। मूत्ररोधार्तिकण्डूभिर्वातबस्तिः स उच्यते॥ (Ca.Si.9/37)

Due to Vidhāraṇat (Mūtravegarodhāt - suppressing the urge of micturition) the Vāta gets aggravated in the Basti (bladder) because of Mūtra dhārayato (withholding the urine) and produces Mūtrarodha (retention of urine), Arti (pain) and Kaṇḍū (itching sensation). This condition is known as Vātabasti.

⊙ वेगं विधारयेद्यस्तु मूत्रस्याकुशलो नरः । निरुणद्धि मुखं तस्य बस्तेर्बस्तिगतोऽनिलः ॥
मूत्रसङ्गो भवेत्तेन बस्तिकुक्षिनिपीडितः । वातबस्तिः स विज्ञेयो व्याधिः कृच्छ्रप्रसाधनः । (Su.U.58/9-10)

When the Akuśala naraḥ (Mūrkha – unwise person) involves in the Vega vidhārana (suppression of urge of micturition), the Vāta located in the Basti (bladder) gets aggravated and causes Basti mukhaṃ niruṇaddhi (obstruction in the orifice of bladder) resulting in Mūtrasaṅga (retention of urine) and Bastikukṣīnipīḍita (severe pain in the bladder and abdomen). This disease is known as Vātabasti and it is Kṛcchraprasādha (Kaṣṭasādhya - difficult to cure).

Uṣṇamāruta or Uṣṇavāta

⊙ उष्मणा सोष्मकं मूत्रं शोषयन् रक्तपीतकम्। उष्णवातः सृजेत् कृच्छ्राद्बस्त्युपस्थार्तिदाहवान्॥ (Ca.Si.9/38)

Due to indulgence in Uṣmaṇā (Pittoṣmaṇā - factors which increases the heat of Pitta in the body) in the Ūṣmaka (hot season), the Vāta gets aggravated along with Pitta and produces Mūtraśoṣayan (dries up the urine), Raktapītaka (reddish or yellowish urine), Sṛjet kṛcchra (urine passed with difficulty) and Bastyupasthitārtidāhavān (pain and burning sensation in the bladder and penis). This condition is known as Uṣṇavāta.

⊙ व्यायामाध्वातपैः पित्तं बस्तिं प्राप्यानिलावृतम् । बस्तिं मेढ्रं गुदं चैव प्रदहन् स्रावयेदधः ॥
मूत्रं हारिद्रमथवा सरक्तं रक्तमेव वा । कृच्छ्रात् प्रवर्तते जन्तोरुष्णवातं वदन्ति तम् ॥ (Su.U.58/22-23)

Due to indulgence in the Vyāyāma (physical exercise), Adhva (long journey on foot) and Ātapa (exposure to the sun), the aggravated Pitta reaches the Basti (bladder) and causes Anilāvṛta (covers up the Vāta) resulting in Basti meḍhra guda pradahan srāvayedadhaḥ (burning sensation in the bladder, penis and rectum during micturition), Mūtraṃ Hāridramathavā saraktaṃ raktameva vā (the urine is turmeric coloured or blood streaked or blood alone) and Kṛcchrāt pravartate (passes with difficulty). This disease is known as Uṣṇavāta.

Mūtraśukra

⊙ प्रत्युपस्थितमूत्रस्तु मैथुनं योऽभिनन्दति । तस्य मूत्रयुतं रेतः सहसा सम्प्रवर्तते ॥
पुरस्ताद्वाऽपि मूत्रस्य पश्चाद्वाऽपि कदाचन । भस्मोदकप्रतीकाशं मूत्रशुक्रं तदुच्यते ॥ (Su.U.58/20-21)

When the person, Pratyupasthitamūtrastu maithunaṃ yoabhinandati (indulges in sexual intercourse during the urge of micturition due to desire), the Retas (semen) suddenly gets ejaculated mixed with Mūtra (urine), which resembles Bhasmodaka (ash water) and it ejaculates before or after micturition. This condition is known as Mūtraśukra.

Vātakuṇḍalikā

⊙ गतिसङ्गादुदावृत्तः स मूत्रस्थानमार्गयोः। मूत्रस्य विगुणो वायुर्भग्नव्याविद्धकुण्डली॥
मूत्रं विहन्ति संस्तम्भभङ्गगौरववेष्टनैः। तीव्ररुङ्मूत्रविट्सङ्गैर्वातकुण्डलिकेति सा। (Ca.Si.9/39-40)

Due to Mūtra gatisaṅga (obstruction in the pathway of the urine), the Viguṇa Vāta (aggravated Vāta) moves upwards and afflicts the Mūtrasthāna (bladder) and Mūtramārga (Mūtravaha srotas) and it becomes Bhagna (Pratihataḥ - broken), Vyāviddha (Vakrī - curved) and Kuṇḍalī (Āvartamānatayā sthita – going round or whirl manner) in its pathway and produces the disease Vātakuṇḍalikā in which the person suffers from Mūtraṃ vihanti (obstruction of urine), Saṃsthambha (stiffness), Bhaṅga (breaking pain), Gaurava (heaviness in the bladder), Veṣṭana (twisting pain), Tīvraruk (severe pain) and Mūtraviṭsaṅga (retention of urine and feces).

⊙ रौक्ष्याद्वेगविघाताद्वा वायुरन्तरमाश्रितः ।मूत्रं चरति सङ्गृह्य विगुणः कुण्डलीकृतः ॥
सृजेदल्पाल्पमथवा सरुजस्कं शनैः शनैः । वातकुण्डलिकां तं तु व्याधिं विद्यात् सुदारुणम्॥ (Su.U.58/5-6)

Due to Raukṣyāt (roughness) or Vegavighātāt (suppression of urge), the Vāta localized in the Bastimadhyama (middle of the bladder) gets aggravated and mixed up with the Mūtrasaṃgṛhya (collected urine) moves like Kūṇḍalīkṛta (whirl or round manner) in the bladder and produces the disease Vātakuṇḍalikā in which the person passes urine Alpamalpa (little by little) or Sarujaskaṃ śanaiḥ śanaiḥ (with pain again and again).

Granthi or Mūtragranthi or Raktagranthi

⊙ रक्तं वातकफाद्दुष्टं बस्तिद्वारे सुदारुणम्। ग्रन्थिं कुर्यात् स कृच्छ्रेण सृजेन्मूत्रं तदावृतम्॥
अश्मरीसमशूलं तं रक्तग्रन्थिं प्रचक्षते। (Ca.Si.9/41-42)

The Rakta gets aggravated by Vāta and Kapha produces Sudāruṇa Granthi (dreadful swelling) in the Bastidvāra (openings of the bladder). Because of this Āvṛta (obstruction), the person suffers from Mūtrakṛcchreṇa (difficulty in urination) and Aśmarīsamaśūla (pain equivalent to calculi). This condition is known as Raktagranthi.

⊙ अभ्यन्तरे बस्तिमुखे वृत्तोऽल्पः स्थिर एव च । वेदनावानति सदा मूत्रमार्गनिरोधनः ॥
जायते सहसा यस्य ग्रन्थिरश्मरिलक्षणः । स मूत्रग्रन्थिरित्येवमुच्यते वेदनादिभिः। (Su.U.58/18-19)

Granthi (swelling) which is Vṛtto (round), Alpa (small) and Sthira (fixed) manifested suddenly in the Abhyantara bastimukha (Bastidvārasyābhyantara - interior side of the orifice of the bladder) produces Vedanāvānati (severe pain), Sadā mūtramārganirodhana (always obstructs the urinary passage) and Aśmarilakṣaṇa (features of calculi). This condition is known as Mūtragranthi.

Viḍghāta

- रूक्षदुर्बलयोर्वातेनोदावृत्तं शकृद्यदा॥ मूत्रस्रोतः प्रपद्येत विट्संसृष्टं तदा नरः।
  विड्गन्धं मूत्रयेत् कृच्छ्राद्विड्विघातं विनिर्दिशेत्॥ (Ca.Si.9/42-43)

When the Śakṛt (faeces) gets occluded by Vāta moves upwards and enters the Mūtravaha srotas in the Rūkṣa (emaciated) and Durbala (weak) person leads to Viṭsaṃsṛṣṭa mūtra (urine mixed with faeces) resulting in Viḍgandha mūtrayet kṛcchrāt (passes urine with faecal odour and difficulty). This condition is known as Viḍghāta or Viḍvighāta.

Bastikuṇḍala

- द्रुताध्वलङ्घनायासादभिघातात् प्रपीडनात्। स्वस्थानाद्बस्तिरुद्वृत्तः स्थूलस्तिष्ठति गर्भवत्॥
  शूलस्पन्दनदाहार्तो विन्दुं विन्दुं स्रवत्यपि। पीडितस्तु सृजेद्धारां संस्तम्भोद्वेष्टनार्तिमान्॥
  बस्तिकुण्डलमाहुस्तं घोरं शस्त्रविषोपमम्। पवनप्रबलं प्रायो दुर्निवारमबुद्धिभिः॥
  तस्मिन् पित्तान्विते दाहः शूलं मूत्रविवर्णता। श्लेष्मणा गौरवं शोफः स्निग्धं मूत्रं घनं सितम्॥
  श्लेष्मरुद्धविलो वस्तिः पित्तोदीर्णो न सिध्यति। अविभ्रान्तबिलः साध्यो न तु यः कुण्डलीकृतः॥
  स्याद्वस्तौ कुण्डलीभूते हृन्मोहः श्वास एव च। (Ca.Si.9/44-49)

Due to Drutādhva (fast walking), Laṅghana (fasting), Āyāsāt (fatigue or stress), Abhighātāt (injury) and Prapīḍanāt (squeezing or pressing), the Svasthānādbasti (bladder in its normal site) gets Udvṛtta (turned up or displaced upwards) and becomes Sthūlastiṣṭati garbhavat (enlarged like gravid uterus) and produces Śūla (colic pain), Spandana (throbbing pain), Dāhārti (burning sensation), Bindu bindu sravat (passes urine drop by drop) and Pīḍitastu sṛjeddhārāṃ (passes urine continuously on pressing or squeezing the bladder by hand etc.) associated with Saṃstambha (stiffness), Veṣṭana (girdle pain) and Arti (pain). This dreadful disease is known as Bastikuṇḍala and it is similar to Śastra (sharp weapon) and Viṣa (poison) in its action. This condition is Pavanaprabala (predominant by Vāta) and it becomes incurable by the unintelligent physicians.

If Pitta is associated, then Dāha (burning sensation), Śūla (colic pain) and Mūtravivarṇatā (discoloration of urine) are manifested and if Kapha is associated, then Gaurava (heaviness), Śopha (swelling) and Snigdhamūtraṃ ghanaṃ sitam (passes cold, thick and slimy urine) are manifested.

If the Kapha gets obstructed in the Bastibila (orifice of the bladder) and Pitta is severely aggravated, then it is Na siddyati (not treatable); in case of Avibhrāntabila (unobstructed orifice of the bladder), then it is Sādhya (curable) and Bastikuṇḍalī causes Hṛnmoha (contraction or oppression of the heart) and Śvāsa (dyspnoea).

Difference between Mūtrāghāta and Mūtrakṛcchra

	Mūtrāghāta	Mūtrakṛcchra
Nirūkti	Mūtra Nirodha or Mūtra śoṣyate and Mūtra pratihanyate	Mūtrasya kṛcchrena mahatā duḥkhena pravṛtti
Doṣa Pradhāna	Vāta pradhāna tridoṣas	Vāta pradhāna tridoṣas
Bheda	13 types	8 types
Nidāna	Veganirodha is the prime cause	Āhāraja Nidāna is the prime cause
Pratyātma Lakṣaṇa	Vibandho balavān and Kṛcchratvamalpa	Kṛcchratvamadhika and Vibandhaścālpa
Modern parlance	Obstructive uropathy	UTI

In Contemporary science,

Mūtrāghāta can be correlated with obstructive uropathy in contemporary science. Obstructive uropathy is a condition characterised by acute or chronic, partial or complete and unilateral or bilateral obstruction in the urinary flow which may be due to structural or functional hindrance. Obstruction can occur at any stage, from the renal tubules to the external urethral meatus, which can lead to increased distension and intraluminal pressure, urinary stasis, urinary tract infection and calculus formation. Acquired and congenital obstructive uropathy such as urethral strictures and meatal stenosis affects all age groups and both sexes. But it is more common in males due to benign prostatic hypertrophy (BPH). (Please refer Topic 11)

MCQ's

1. Mūtrāghāta causes

   a) Mūtrāvarodha b) Mūtra śoṣyate
   c) Mūtra pratihanyate d) All of the above

2. According to Caraka, Mūtrāghāta is of .... types

   a) 4 b) 8
   c) 12 d) 13

3. According to Suśruta, Mūtrāghāta is of .... types

   a) 4 b) 8
   c) 12 d) 13

4. According to the Caraka, which of the following types of Mūtrāghāta is NOT mentioned
    a) Mūtrajaṭhara b) Mutrotsaṅga
    c) Mūtrātītā d) Mūtraśukra
5. According to the Suśruta, which of the following types of Mūtrāghāta is NOT mentioned
    a) Mūtrajaṭhara b) Viḍvighāta
    c) Mūtrātītā d) Mūtraśukra
6. बस्तिकुक्षिनिपीडितः is the feature of
    a) Mūtraukasāda b) Mūtrātītā
    c) Vātabasti d) Uṣṇavāta
7. भस्मोदकप्रतीकाशं is the feature of
    a) Mūtrajaṭhara b) Mūtragranthi
    c) Mūtrātītā d) Mūtraśukra
8. अश्मरीसमशूलं is the feature of
    a) Mūtrajaṭhara b) Mūtragranthi
    c) Mūtrātītā d) Mūtraśukra
9. बिन्दुं बिन्दुं स्रवत्यपि is the feature of
    a) Uṣṇavāta b) Vātabasti
    c) Vātakuṇḍalikā d) Bastikuṇḍala
10. बस्त्युपस्थार्तिदाहवान् is the feature of
    a) Uṣṇavāta b) Vātabasti
    c) Vātakuṇḍalikā d) Bastikuṇḍala

Answers Keys

1	d	2	d	3	c	4	d	5	b
6	c	7	d	8	b	9	d	10	a

Short Answer Questions

1. Describe different types of Mūtrāghāta
2. Write the difference between Mūtrāghāta and Mūtrakṛcchra

◆

TOPIC 11

Common Urinary Diseases

Introduction

Urinary diseases encompass a variety of conditions that affect the organs responsible for the production, storage, and elimination of urine, specifically the kidneys, ureters, bladder, and urethra. These diseases can manifest in numerous ways, ranging from infections and inflammatory responses to structural abnormalities and malignancies. Common urinary diseases include urinary tract infections (UTIs), kidney stones, polycystic kidney disease, nephrotic syndrome, nephritic syndrome, as well as acute and chronic kidney disease. Additionally, benign prostatic hyperplasia (BPH) and bladder cancer are significant concerns within this field. This chapter aims to provide a comprehensive analysis of selected conditions, focusing on urinary tract infections, Prostatomegaly, nephrotic syndrome, nephritic syndrome, acute kidney injury and chronic kidney disease.

URINARY TRACT INFECTIONS

Introduction

A urinary tract infection (UTI) is an infection that affects any part of the urinary tract such as urethra (urethritis), bladder (cystitis), prostate (prostatitis), ureters (ureteritis) and kidneys (pyelonephritis). But the term urinary tract infection usually refers to cystitis and pyelonephritis. Most commonly, it affects the bladder (cystitis), which is also known as uncomplicated UTI and less commonly it affects the kidneys (pyelonephritis) but more serious than cystitis.

Cystitis

Cystitis is defined as an inflammation of the urinary bladder and is recognized as the most prevalent type of lower urinary tract infection, caused by microorganisms. This condition occurs more frequently in women compared to men, primarily due to the shorter length of the female urethra and the presence of bacteria in the periurethral area. The most common bacterial pathogen associated with cystitis is Escherichia coli; however, other notable pathogens include Proteus mirabilis, Klebsiella pneumoniae, and Staphylococcus saprophyticus.

In the year 2019, it was reported that over 404.6 million individuals worldwide were affected by urinary tract infections (UTIs). India documented the highest incidence, with approximately 100.8 million cases, leading to 55,558 fatalities and contributing to 1.59 million disability-adjusted life years (DALYs) related to these infections.

Cystitis is classified into two distinct types: acute uncomplicated cystitis and complicated cystitis, distinguished by the duration of the infection.

- **Acute uncomplicated cystitis** primarily affects women, with several factors identified as increasing the risk of infection. These factors include sexual intercourse, the utilization of spermicides, a history of previous urinary tract infections (UTIs), a strong familial predisposition to UTIs in first-degree female relatives, and the pre-menopausal state.
- **Complicated cystitis** is predominantly seen in men. The risk factors associated with this condition encompass a history of childhood UTIs, an immuno-compromised status, pregnancy, metabolic disorders such as diabetes mellitus, urologic abnormalities including nephrolithiasis, the presence of urinary stents, strictures, indwelling urinary catheters, neurogenic bladder, and polycystic kidney disease.

Pathophysiology

The pathophysiology of cystitis is characterized by the adherence of bacteria to the bladder wall, which facilitates their invasion and colonization of the uroepithelial cells. This bacterial invasion precipitates an inflammatory response marked by the release of cytokines and other inflammatory mediators. Consequently, individuals experience the hallmark symptoms of cystitis, which include frequent and painful urination, urgency, and suprapubic pain. The resulting inflammation may increase the sensitivity and irritation of the bladder, leading to increased discomfort and an urgent need to urinate even when the bladder is not distended. Additional factors, such as urinary stasis, inadequate hygiene, and catheterization, further increase the susceptibility of individuals to cystitis.

Clinical features:

The clinical features of cystitis include burning pain in the urethra during micturition (dysuria), urinary frequency and urgency, suprapubic pain, dark and cloudy urine with

unpleasant odour and occasionally haematuria. Systemic symptoms such as fever, chills, malaise are usually mild or absent.

Complications:

The potential complications are Pyelonephritis, renal or perinephric abscess formation, renal vein thrombosis, sepsis, acute renal failure and emphysematous-pyelonephritis

Diagnosis:

Diagnosis is based on the urinalysis and urine culture. Urinalysis shows the presence of pyuria i.e. > 10 WBC's or leukocytes in an unspun urine. Positive urine dipstick test for leukocyte esterase or nitrite indicates the presence of Enterobacteriaceae. Urine culture is helpful to identify the pathogen and for determining antimicrobial susceptibility profiles. Other investigations such as plain x-ray abdomen, excretion urography and ultrasonography are done to evaluate the other causes such as congenital anomalies, calculi and other abnormalities.

Nephritis

Nephritis is characterized by inflammation of the kidneys, which may be classified as either acute or chronic. This condition can arise from a variety of factors, including infections, autoimmune diseases, and certain medications or toxins. The global prevalence of nephritis is estimated to affect approximately 2-5% of the population, with an incidence ranging from 2 to 10 cases per 100,000 individuals annually. In India, studies indicate that nephritis impacts approximately 3-6% of the population, with a notably higher prevalence in urban regions (4.3%) compared to rural areas (2.6%). The condition is more prevalent among males than females, particularly in the group aged 20-40 years.

Nephritis can be categorized into four primary types: glomerulonephritis, interstitial nephritis, pyelonephritis, and lupus nephritis.

- **Glomerulonephritis:** This type is characterized by the inflammation of the glomeruli and is further classified into primary and secondary forms based on etiology. Primary glomerulonephritis is often idiopathic, while secondary forms may result from various infections (including streptococcal, mycoplasma, salmonella, staphylococcus, Plasmodium falciparum, Plasmodium malariae, cytomegalovirus, Epstein-Barr virus, hepatitis, and varicella) as well as systemic diseases such as Goodpasture syndrome, systemic lupus erythematosus (SLE), hemolytic uremic syndrome, polyarteritis nodosa, and Alport syndrome.
- **Interstitial nephritis:** This condition involves inflammation of the interstitial tissues between the renal tubules and is also known as tubulointerstitial nephritis. Causes include a range of infections such as E. coli, HIV, leptospirosis, syphilis, toxoplasmosis, leishmaniasis, histoplasmosis, and coccidioidomycosis. Medications

include antibiotics, anticonvulsants, non-steroidal anti-inflammatory drugs (NSAIDs), proton pump inhibitors, and acyclovir, as well as systemic conditions like sarcoidosis, Sjogren's syndrome, IgG4-related disease, and systemic lupus erythematosus can also contribute to this form of nephritis.

- **Pyelonephritis:** This type of nephritis results from a urinary tract infection that reaches to the renal pelvis (pyelum). It is primarily associated with gram-negative bacteria, particularly Escherichia coli, although other organisms such as Proteus, Klebsiella, and Enterobacter may also be included.
- **Lupus nephritis:** This is an inflammation of the kidneys associated with the autoimmune disease known as systemic lupus erythematosus (SLE).

Pathophysiology

The pathophysiology of nephritis typically begins with an initial trigger, which may include an infection, an autoimmune disorder, or exposure to toxins. This inciting event leads to the activation of an immune response, during which T cells and macrophages are mobilized to release pro-inflammatory cytokines. Consequently, this results in inflammation and oxidative stress within the kidneys. The glomeruli, which are the tiny blood vessels responsible for filtration, become damage, permitting proteins and blood to enter the urine. Additionally, the inflammatory process adversely affects the renal tubules and interstitium, thereby disrupting electrolyte balance and fluid regulation. As the condition progresses, fibrosis and scarring occur, ultimately impairing kidney function and increasing the risk of chronic kidney disease or end-stage renal failure. Although the specific mechanisms differ depending on the type of nephritis such as acute post-streptococcal glomerulonephritis, lupus nephritis, or IgA nephropathy, the common outcome is renal dysfunction, which may advance to kidney failure.

Clinical features:

The common symptoms of nephritis include back pain, a burning sensation during micturition, puffiness of the face, and oedema in the legs and feet. Other signs may involve high blood pressure, tachycardia, abdominal pain, headache, vomiting, fever, and a decreased amount of urine (oliguria). The urine may exhibit a dark, smoky, or reddish-brown appearance, often containing albumin and blood.

Complications:

The potential complications of nephritis include hypertensive encephalopathy, acute left heart failure, pulmonary edema, and renal failure.

Diagnosis:

Diagnosis is based on the clinical evaluation, urinalysis, renal function test, imaging studies and kidney biopsy. Clinical evaluation reveals that oedema first appears on the face and then spreads to all over the body. Urinalysis shows the presence of albumin, red blood cells and epithelial casts. Renal function test demonstrates the elevated levels of blood urea and creatinine. Imaging studies such as USG, CT scan and X-ray helps to identify the blockage and inflammation. Kidney biopsy helps to confirm the diagnosis.

PROSTATOMEGALY

Introduction

Prostatomegaly is the enlargement of the prostate gland caused by various factors, often associated with benign prostatic hyperplasia (BPH). It is characterized by excessive growth of prostatic tissue, leading to bladder outlet obstruction, and it commonly affects men over 40. A 2017 study reported that the prevalence of BPH in India was 6.7-14%. The exact cause of BPH is unknown, but the hormonal effects of testosterone on prostate tissues may play a role in cellular proliferation. Other risk factors, such as metabolic syndrome, obesity, hypertension, and genetic factors, also contribute to the development of BPH.

Pathophysiology

The pathophysiology of Prostatomegaly involves the enlargement of the prostate gland, primarily due to hormonal factors. Dihydrotestosterone (DHT), a metabolite of testosterone, plays a critical role in stimulating the growth of prostate cells. This process begins with the conversion of testosterone into DHT by the enzyme 5α-reductase. Subsequently, DHT binds to androgen receptors within the prostate, resulting in increased cellular proliferation, particularly in the transition zone of the gland. This results in the formation of large, distinct nodules. Chronic inflammation is also believed to contribute to the condition. As the prostate enlarges, it compresses the urethra, causing lower urinary tract symptoms (LUTS) such as increased urinary frequency, urgency, nocturia, and difficulty initiating urination.

Clinical features:

Benign prostatic hyperplasia is the most common cause of lower urinary tract symptoms, which can be divided into storage and voiding symptoms. Storage symptoms are increased urine frequency, urgency, and nocturia (waking at night to micturate). In contrast, voiding symptoms are weak urinary stream, urinary hesitancy (delay between trying to micturate and the actual beginning of flow), straining to void, and prolonged micturition.

Diagnosis:

The diagnosis is based on digital rectal examination, urinalysis, urine culture, prostate-specific antigen level, and ultrasonography. A digital rectal examination reveals a smooth, enlarged prostate. Urinalysis and culture are done to rule out other causes, such as infection.

The PSA level helps detect prostatic cancer, and the USG scan demonstrates the high residual volume of urine.

NEPHROTIC SYNDROME

Introduction

Nephrotic syndrome is a clinical syndrome characterized by severe proteinuria (>3.5g/24 hours), often associated with hypoalbuminemia (<30g/L), oedema, hyperlipidemia and various complications. It occurs at any age but is more common in children, mostly between 1 and 4 years of age, and boys are affected more often than girls. It is caused by an increased permeability through the damaged basement membrane in the renal glomerulus, especially infectious or thrombo-embolic, which allows proteins such as albumin, antithrombin or immunoglobulins to pass through the cell membrane and appear in the urine. Globally, the annual incidence of nephrotic syndrome ranges from 1.2 to 16.9 per 100,000 children. In India, the prevalence of idiopathic nephrotic syndrome varies from 12-16 per 100,000 children.

The primary causes of nephrotic syndrome are minimal-change glomerulonephritis, focal glomerulonephritis, membranous nephropathy and hereditary nephropathies. Secondary causes of nephrotic syndrome are diabetes mellitus, lupus erythematosus, renal amyloidosis, drugs like NSAIDs, gold salts, penicillamine, etc., renal vein thrombosis, infections like HIV, hepatitis B virus, hepatitis C, cytomegalovirus, toxoplasmosis, etc., and preeclampsia.

Pathophysiology

The pathophysiology of nephrotic syndrome is characterized by damage to the glomerular filtration barrier, which permits the leakage of albumin and other proteins into the urine. This damage may arise from various etiologies, including minimal change disease, focal segmental glomerulosclerosis, membranous nephropathy, and diabetic nephropathy. The loss of negatively charged albumin and other proteins disturbs the electrostatic forces within the glomerulus, resulting in increased permeability and an exacerbation of proteinuria. As albumin levels decrease, the oncotic pressure in the blood vessels diminishes, prompting a fluid shift from the vascular compartment to the interstitial spaces, thereby causing oedema. In response to hypoalbuminemia, the liver compensates by increasing lipoprotein synthesis, which leads to hyperlipidemia. Additionally, the kidney's increased reabsorption of proteins and lipids may contribute to further renal damage and fibrosis, thereby perpetuating the cycle of the disease.

Clinical features:

The cardinal clinical features of nephrotic syndrome are generalized oedema and puffiness of eyelids. Other features include pitting oedema over legs and feet, tachycardia, anaemia, loss of appetite, frothy urine, malaise and generalized weakness. In severe cases, ascites, arthralgia and breathlessness due to pleural effusion can be seen.

Diagnosis:

Diagnosis is based on the clinical presentation, urinalysis, 24 hours urine test, blood investigation and renal biopsy. Clinically, massive generalized oedema and albuminuria on urinalysis demonstrates the nephrotic syndrome. Urine samples over 24 hours shows proteinuria (>3g/day) and is diagnostic. Blood tests demonstrates low level of albumin (<2.5mg/ dl) and increased level of total cholesterol and triglycerides. Renal biopsy helps to confirm the histological diagnosis.

NEPHRITIC SYNDROME

Introduction

Nephritic syndrome is defined as a renal condition characterized by inflammatory damage to the glomerular capillaries. This syndrome may arise from an underlying kidney disease or develop as a consequence of systemic disorders. A range of etiological factors can contribute to its occurrence, including autoimmune conditions, hereditary factors, and infectious diseases such as acute proliferative glomerulonephritis (which includes both post-infectious and infection-associated types), crescentic glomerulonephritis, and proliferative lupus glomerulonephritis. Nephritic syndrome frequently presents in various forms of proliferative glomerulonephritis (GN). The global incidence of nephritic syndrome is estimated to be between 2 and 10 cases per 100,000 individuals per year, while in India, the estimated incidence is approximately 5 to 7 cases per 100,000 individuals annually.

Pathophysiology

The pathophysiology of nephritic syndrome involves an immune-mediated response, which is frequently initiated by infections, autoimmune disorders, or vasculitic conditions. This immune response leads to the deposition of immune complexes or the activation of complement proteins within the glomerular basement membrane, resulting in damage to the capillary walls. Consequently, this damage increases the permeability of the glomeruli, allowing the leakage of proteins and blood into the urine. The resultant injury triggers an inflammatory response and the proliferation of glomerular cells, further impairing renal function. The affected glomeruli exhibit thickening of the basement membrane, increased cellularity, and infiltration by leucocytes. Furthermore, activation of the coagulation cascade may occur, leading to fibrin deposition and additional renal injury. These combined effects of inflammation, immune complex deposition, and cellular proliferation disrupt the selective filtration barrier, manifesting in haematuria, proteinuria, hypertension, and diminished renal function, which are characteristic features of nephritic syndrome. The severity of the syndrome is contingent upon the extent and nature of the glomerular damage.

Clinical features:

The clinical manifestation of nephritic syndrome includes Haematuria, pyuria, hypertension, oliguria (reduced urine output), periorbital, and pedal oedema. This condition leads to a sudden onset of the presence of red blood cell (RBC) casts and blood cells, as well as varying levels of proteinuria and white blood cells in the urine.

Diagnosis:

Diagnosis is based on the clinical presentation, urinalysis, kidney biopsy and serological tests. Clinical indicators encompass pallor, anemia, elevated blood pressure, jugular venous pressure (JVP) distention, pitting edema, chest auscultation revealing crackles, and an increase in red blood cells (RBCs) per high power field (>5) along with acanthocytes and dysmorphic RBCs. Furthermore, the presence of red blood cell (RBC) casts and, in some cases, white blood cell (WBC) casts, as well as proteinuria (less than 3.5 g/day) observed in urinalysis, indicate the possibility of Nephritic syndrome. Blood tests typically reveal elevated creatinine levels and reduced glomerular filtration rate (GFR). A kidney biopsy is performed to confirm the histological diagnosis. In addition, serological testing, including ANA, Serum C3 and C4 Complement Levels, ASO titers, Anti-neutrophil cytoplasmic antibodies (ANCA), Anti-dsDNA Antibodies, Anti-glomerular Basement Membrane Antibodies, Hepatitis B Surface Antigen, HCV Antibodies, Serum Protein Electrophoresis, Serum Immunofixation, and Rheumatoid Factor, is conducted to exclude underlying immunological renal diseases.

ACUTE KIDNEY INJURY (AKI)

Introduction

Acute kidney injury (AKI), formerly known as acute renal failure is characterized by a sudden and often reversible reduction in kidney function, as measured by glomerular filtration rate (GFR). A study carried out in southern India revealed an AKI prevalence of approximately 8.36 cases per 1,000 individuals, evidencing a male preponderance (72.7%) with a median age of 56 years. Community-Acquired AKI (CA-AKI) constituted 35% of the cases, while Hospital-Acquired AKI (HA-AKI) accounted for 65%. The primary etiologies for CA-AKI were chronic kidney disease (CKD) at 23% and sepsis at 22.4%, whereas HA-AKI was principally associated with sepsis (49.2%) and CKD (11.9%). The etiology of AKI can be categorized as prerenal, intrarenal and postrenal

- **Prerenal AKI** results from inadequate renal perfusion and is attributable to hypovolemia (e.g., from hemorrhage or severe burns), hypotension due to decreased cardiac output (e.g., cardiogenic shock or massive pulmonary embolism), systemic vasodilatation (e.g., septic shock or anaphylaxis), renal vasoconstriction (caused by NSAIDs, calcineurin inhibitors, hepatorenal syndrome), and glomerular efferent arteriolar vasodilation (resulting from drugs such as angiotensin receptor blockers (ARB), ACE Inhibitors, etc.).
- **Intrarenal AKI** is characterised by intrinsic renal disease or damage affecting blood vessels, glomeruli, tubules, or interstitial tissue. Causes encompass acute tubular necrosis, acute glomerulonephritis, acute interstitial nephritis, and intratubular obstruction.
- **Postrenal AKI** is distinguished by obstructive nephropathy due to extrinsic compression (e.g., prostatic hypertrophy, carcinoma), intrinsic obstruction (e.g., calculi, tumors, clots, strictures), and decreased function (as seen in conditions like neurogenic bladder).

Pathophysiology

The pathophysiology of AKI involves a complex interplay of vascular, tubular, and inflammatory mechanisms. Initially, a decrease in renal blood flow occurs due to vasoconstriction or diminished cardiac output, leading to hypoxia and subsequent tubular injury. This hypoxic state activates an inflammatory response, resulting in the release of cytokines and chemokines that recruit leukocytes and exacerbate tissue damage. Consequently, tubular cells may experience necrosis or apoptosis, releasing cellular debris that can obstruct tubular lumens. The kidneys capacity to effectively reabsorb and excrete waste products is compromised, resulting in the accumulation of toxins, electrolyte imbalances, and fluid overload. Furthermore, increased permeability of the renal tubules allows for the backflow of filtrate into the bloodstream. As AKI progresses, the kidneys self-regulatory mechanisms become ineffective, leading to a further decline in glomerular filtration rate (GFR) and potentially irreversible damage if left untreated. The severity of AKI is classified into stages, ranging from mild (Stage 1) to severe (Stage 3), with higher stages correlating with increased morbidity and mortality.

The staging criteria for Acute Kidney Injury (KDIGO 2012) are based on the serum creatinine level or the amount of urine output

- **Stage 1:** An increase in serum creatinine ≥0.3mg/dl or 1.5-1.9 times baseline and urine output is <0.5ml/kg/hour for 6-12 hours
- **Stage 2:** An increase in serum creatinine 2-2.9 times baseline and urine output is <0.5ml/ kg/hour for ≥ 12 hours
- **Stage 3:** An increase in serum creatinine ≥4.0mg/dl or ≥3 times baseline and urine output is <0.3ml/kg/hour for ≥ 24 hours or anuria for ≥12 hours. Renal replacement therapy is indicated in this stage.

Clinical features:

Decreased urine output (oliguria <400ml/day), pedal oedema, weakness, shortness of breath, confusion, nausea, lethargy and arrhythmias (abnormal heart rhythms). In severe cases, chest pain, pericardial tamponade, pleural effusion, seizures or coma may occur.

Diagnosis:

Diagnosis is based on Kidney Disease: Improving Global Outcomes (KDIGO) criteria in 2012. AKI is diagnosed with any of the following

- Increase in serum creatinine by ≥0.3 mg/dl within 48 hours; or
- Increase in serum creatinine to 1.5 times or more than the baseline of the prior 7 days or
- Urine volume is less than 0.5 ml/kg/h for 6 hours.

CHRONIC KIDNEY DISEASE (CKD)

Introduction

Chronic Kidney Disease (CKD) is clinically defined by the presence of kidney damage or an estimated glomerular filtration rate (eGFR) of less than 60 ml/min per 1.73 square meters, persisting for duration of 3 months or more. CKD represents a significant risk for progression to end stage renal disease (ESRD), necessitating renal replacement therapy. The reported prevalence of CKD in various regions of India ranges from less than 1% to 13%. Recent data from the International Society of Nephrology's Kidney Disease Data Center Study has indicated a prevalence of 17%.

The primary etiologies of chronic kidney disease (CKD) include diabetes mellitus, hypertension, primary glomerulonephritis, chronic tubulointerstitial nephritis, polycystic kidney diseases, hereditary or cystic diseases, secondary glomerulonephritis or vasculitis, and plasma cell dyscrasias or neoplasms. CKD may emerge from pathological processes falling into three categories: prerenal (associated with decreased renal perfusion pressure), intrinsic renal (pertaining to pathology of the vessels, glomeruli, or tubules-interstitium), or postrenal (obstructive).

Prerenal CKD manifests in patients with chronic heart failure or cirrhosis leading to persistently decreased renal perfusion, increase susceptibility to recurrent episodes of intrinsic kidney injury such as acute tubular necrosis (ATN), consequently resulting in the gradual decline of renal function.

Intrinsic CKD develops in patients with chronic renal vascular disease (nephrosclerosis, renal artery stenosis), intrinsic glomerular disease (nephritic or nephrotic), and chronic tubulointerstitial disease (polycystic kidney disease).

Postrenal CKD is characterized by obstructive nephropathy due to prostatic disease, nephrolithiasis, or abdominal/pelvic tumors exerting mass effect on ureters. Common underlying causes include retroperitoneal fibrosis and chronic ureteral obstruction.

Pathophysiology:

The pathophysiology of chronic kidney disease (CKD) involves a complex interplay of factors that start with initial kidney injury due to diabetes, hypertension, or glomerulonephritis. This injury triggers a cascade of inflammatory responses, oxidative stress, and fibrosis, which damages the glomeruli, tubules, and blood vessels. The decline in kidney function results in a decreased glomerular filtration rate (GFR), leading to the retention of waste products, electrolyte imbalances, and fluid overload. The impaired kidneys struggle to regulate electrolytes, acid-base balance, and blood pressure, causing complications like hyperkalemia, metabolic acidosis, and hypertension. Additionally, CKD affects vitamin D activation, leading to secondary hyperparathyroidism, bone disease, and mineral metabolism disorders.

Clinical features:

The early stages of CKD are usually asymptomatic despite elevated blood urea and creatinine. Nocturia, foamy or bubbly and cloudy or tea-coloured urine, oedema, hypertension, fatigue, shortness of breath, anorexia, nausea, vomiting, thirst, bad taste in the mouth, weight loss, persistent pruritus, yellowish brown pigmentation, muscle twitching or cramping and hyperreflexia. In severe cases, pericarditis, gastrointestinal ulceration and bleeding, anaemia, cardiac failure or coronary artery disease, dependant oedema and seizure may occur.

Diagnosis:

Diagnosis is based on Kidney Disease: Improving Global Outcomes (KDIGO) criteria established in 2012; chronic kidney disease (CKD) is classified into six distinct groups based on the glomerular filtration rate (G1 to G5, with G3 further subdivided into 3a and 3b). Additionally, this classification system incorporates staging determined by three levels of albuminuria (A1, A2, and A3). Each stage of CKD is further delineated by the urinary albumin-creatinine ratio, measured in (mg/g) or (mg/mmol), extrapolated from an early morning "spot" urine sample.

- **G1:** Normal or high GFR (≥ 90mL/min/1.73 m^2) along with persistent albuminuria or those with abnormal imaging studies.
- **G2:** mildly decreased GFR 60 to 89 mL/min/1.73 m^2
- **G3a:** mildly to moderately decreased GFR 45 to 59 mL/min/1.73 m^2.
- **G3b:** moderately to severely decreased GFR 30 to 44 mL/min/1.73 m^2. Nocturia and polyuria are the initial features appearing at this stage.

- **G4:** severely decreased GFR 15 to 29 mL/min/1.73 m². Overt uremic symptoms like loss of appetite, nausea, anaemia and recurrent infections occur during this stage.
- **G 5:** GFR < 15 mL/min/1.73 m². At this stage, dialysis or renal replacement therapy is required.

The three levels of albuminuria include an albumin-creatinine ratio (ACR)

- **A1:** ACR less than 30 mg/gm (less than 3.4 mg/mmol)
- **A2:** ACR 30 to 299 mg/gm (3.4 to 34 mg/mmol)
- **A3:** ACR greater than 300 mg/gm (greater than 34 mg/mmol).

The enhanced classification of chronic kidney disease (CKD) has proven advantageous in identifying prognostic markers associated with diminished kidney function and heightened albuminuria. Nevertheless, a drawback of employing classification systems is the potential for over diagnosis of CKD, particularly in older people.

MCQ's

1. **What part of the urinary tract is most commonly affected by UTIs**
   a) **Urethra** b) Bladder
   c) **Prostate** d) Kidneys
2. **Which bacterial pathogen is most commonly associated with cystitis**
   a) **Staphylococcus saprophyticus** b) Klebsiella pneumoniae
   c) **Escherichia coli** d) Proteus mirabilis
3. **Which type of cystitis is predominantly seen in men**
   a) **Acute uncomplicated cystitis** b) Complicated cystitis
   c) **Ulcerative cystitis** d) Hemorrhagic cystitis
4. **What is a hallmark symptom of cystitis**
   a) **Jaundice** b) Frequent and painful urination
   c) **Chronic cough** d) Skin rash
5. **What is a potential complication of cystitis**
   a) **Chronic liver disease** b) Renal vein thrombosis
   c) **Diabetes** d) Asthma

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6. Which type of nephritis involves inflammation of the glomeruli?
   a) Interstitial nephritis b) Pyelonephritis
   c) Glomerulonephritis d) Lupus nephritis
7. What is a common bacterial cause of pyelonephritis
   a) Streptococcus b) Escherichia coli
   c) Staphylococcus aureus d) Mycoplasma
8. What potential complication can arise from nephritis
   a) Pulmonary oedema b) Chronic liver disease
   c) Asthma d) Diabetes
9. What hormone is crucial in the pathophysiology of BPH
   a) Estrogen b) Progesterone
   c) Dihydrotestosterone (DHT) d) Cortisol
10. What enzyme converts testosterone into DHT
    a) 5α-reductase b) Aromatase
    c) Lipase d) Amylase
11. Which clinical feature is a voiding symptom of BPH
    a) Urgency b) Nocturia
    c) Weak urinary stream d) Increased urinary frequency
12. What does a digital rectal examination reveal in BPH
    a) Rough, irregular prostate b) Smooth, enlarged prostate
    c) Ulcerated prostate d) Shrunken prostate
13. What characterizes Nephrotic Syndrome
    a) Severe proteinuria (>3.5g/24 hours) b) Low blood pressure
    c) High blood glucose levels d) Normal urine output
14. What clinical feature is characteristic of nephritic syndrome
    a) Chronic cough b) Haematuria and pyuria
    c) Joint pain d) Skin rash
15. What is a common complication of nephritic syndrome
    a) Diabetes b) Hypertensive encephalopathy
    c) Chronic liver disease d) Asthma

16. What characterizes Acute Kidney Injury
    a) Slow, irreversible reduction in kidney function
    b) Sudden, often reversible reduction in kidney function
    c) Chronic kidney disease progression
    d) Stable kidney function
17. What clinical feature is common in AKI
    a) Chronic cough
    b) Decreased urine output (oliguria <400ml/day)
    c) Joint pain
    d) Skin rash
18. What category of CKD is associated with obstructive nephropathy
    a) Prerenal CKD
    b) Intrinsic CKD
    c) Postrenal CKD
    d) Chronic CKD
19. How is Stage 3a CKD classified
    a) GFR ≥ 90 mL/min/1.73 m2
    b) GFR 60 to 89 mL/min/1.73 m2
    c) GFR 45 to 59 mL/min/1.73 m2
    d) GFR 15 to 29 mL/min/1.73 m2
20. What is a potential complication of CKD
    a) Chronic liver disease
    b) Hyperkalemia
    c) Asthma
    d) Diabetes

Answers Keys

1	b	2	c	3	b	4	b	5	b	6	c	7	b	8	a	9	c	10	a
11	c	12	b	13	a	14	b	15	b	16	b	17	b	18	c	19	c	20	b

Short Answer Questions

1. Explain Urinary tract infection
2. Explain Prostatomegaly
3. Explain Nephrotic syndrome
4. Explain Nephritic syndrome
5. Explain Acute kidney injury
6. Explain chronic kidney disease

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TOPIC 12

Hikkā

Introduction

Hikkā, a disorder of Prāṇavaha srotas, may manifest as an independent disease or as a symptom of other disorders. Hikkā is characterized by a disturbance in the Kapha and Vāta doṣas, originating from the site of Pitta. Unlike other diseases, Hikkā can lead to Prāṇāśu nikuntataḥ (immediate death) and is typically evident in the terminal phases of life, irrespective of the presence of other disease conditions. If Hikkā is not treated properly, it can be as destructive to the patient as Āśīviṣa (snake poison). Hidhmā is a synonym of Hikkā.

Nirūkti and Paribhāṣā of Hikkā

- हिक्कातेऽनयेति हिक्का:॥ (SKD)

  That which produces a spasmodic sound in the throat is called Hikkā.
- हिक्कति कूजत्यनया हिक्का:॥ (SKD)

  That which produces an indistinct sound is called Hikkā.
- हिक्कयते हिनस्तोति वा॥ (SKD)

  That which kills or injures the person is called Hikkā.
- हिगिति कृत्वा कायति शब्दायते इति (Ma.M.N.12/3)
- तत्र हिगिति कायति शब्दं करोतीति हिक्का (Ca.Ca.Ci.17/27-30)

  The disease which originates out of breath with 'Hik Hik' sound is called Hikkā.
- हिक्कास्वरुपं तु घोषवत्त्वेन प्रतिपादितं॥ (Āḍ.Sā.P.7/25)

  It is stated that the nature of Hikkā is producing sound.

- ⦿ मुहुर्मुहुर्वायुरुदेति सस्वनो यकृत्प्लीहान्त्राणि मुखादिवाक्षिपन् ।
  स घोषवानाशु हिनस्त्यसून् यतस्ततस्तु हिक्केत्यभिधीयते बुधैः (M.N.12/3)
- ⦿ मुहुर्मुहुर्वायुरुदेति सस्वनो यकृत्प्लीहान्त्राणि मुखादिवाक्षिपन् ।
  स घोषवानाशु हिनस्त्यसून् यतस्ततस्तु हिक्केति भिषग्भिरुच्यते ॥ (Su.U.50/6)

When Vāyu repeatedly and forcefully brings out a specific sound 'Hik' through the mouth by pulling Yakṛt (liver), Plīha (spleen) and Antrāṇi (intestines), and is fatal in nature is called Hikkā by the physicians

Sāmānya Nidāna of Hikkā

- ⦿ रजसा धूमवाताभ्यां शीतस्थानाम्बुसेवनात्। व्यायामाद्ग्राम्यधर्माध्वरूक्षान्नविषमाशनात्॥
  आमप्रदोषादानाहाद्रौक्ष्यादत्यपतर्पणात्। दौर्बल्यान्मर्मणो घाताद्द्वन्द्वाच्छुद्ध्यतियोगतः॥
  अतीसारज्वरच्छर्दिप्रतिश्यायक्षतक्षयात्। रक्तपित्तादुदावर्ताद्विसूच्यलसकादपि॥
  पाण्डुरोगाद्विषाच्चैव प्रवर्तेते गदाविमौ। निष्पावमाषपिण्याकतिलतैलनिषेवणात्॥
  पिष्टशालूकविष्टम्भि विदाहिगुरुभोजनात्। जलजानूपपिशितदध्यामक्षीरसेवनात्॥
  अभिष्यन्द्युपचाराच्च श्लेष्मलानां च सेवनात्। कण्ठोरसः प्रतीघाताद्विबन्धैश्च पृथग्विधैः॥ (Ca.Ci.17/11-16)

Rajodhūmavātābhyāṃ (exposure to dust, smoke and winds), Śītasthānāmbusevanāt (residing in cold environment and intake of cold water), Vyāyāma (excessive exercise), Grāmyadharmā (excessive sexual intercourse), Adhva (walking long distances), Rūkṣānna (intake of non-unctuous food), Viṣamāśana (irregular dietary habits), Āmapradoṣā (afflicted with Āma), Ānāha (abdominal distention and constipation),Raukṣyāt (non- unctuousness), Apatarpaṇa (excessive fasting), Daurbalya (weakness), Marmaṇoghāta (injury to the vital parts), Dvandvā (use of cold and hot substances or wholesome and unwholesome substances), Śuddhāyatiyogataḥ (excessive purificatory procedures) and also due to complications of diseases such as Atisāra, Jvara, Chardi, Pratiśyāya, Kṣata, Kṣaya, Raktapitta, Udāvarta, Visūcī, Alasaka, Pāṇḍuroga ad Viṣa. And also, the disease Hikkā and Śvāsa get manifested due to intake of Niṣpāva, Māṣa, Piṇyāka, Tilataila, Piṣṭaśālūka (pastry and stem of lotus), Viṣṭambhividāhigurubhojanāt (intake of food substances which are constipated, producing burning sensation and heavy), Jalajānūpapiśitadadhyāmakṣīrasevanāt (intake of meat of aquatic and marshy animals, curds and unboiled milk), Abhiṣyandī (channels blocking substances), Śleṣmalānāṃ ca sevanat (use of Kapha aggravating factors); Kaṇṭhorasaḥ pratighāta (injury to throat and chest) and Vibandhaśca pṛthagvidaiḥ (various types of constipation)

- ⦿ विदाहिगुरुविष्टम्भिरूक्षाभिष्यन्दिभोजनैः। शीतपानाशनस्थानरजोधूमातपानिलैः॥
  व्यायामकर्म भाराध्ववेगाघातापतर्पणैः । हिक्का श्वासश्च कासश्च नृणां समुपजायते ॥ (M.N.12/1-2)
- ⦿ विदाहिगुरुविष्टम्भिरूक्षाभिष्यन्दिभोजनैः। शीतपानाशनस्थानरजोधूमानिलानलैः ॥
  व्यायामकर्म भाराध्ववेगाघातापतर्पणैः । आमादोषाभिघातस्त्रीक्षयदोषप्रपीडनैः ॥
  विषमाशनाध्यनशनैस्तथा समशनैरपि । हिक्का श्वासश्च कासश्च नृणां समुपजायते ॥ (Su.U.50/3-5)

Indulging in food which are Vidāhi (producing burning sensation), Guru (heavy), Viṣṭambhi (constipation), Rūkṣa (non-unctuous) and Abhiṣyandi bhojanaḥ (channels blocking substances), Śītapānāsanasthāna (intake of cold water and exposure to cold environment), Rajodhūmānillānalaiḥ (exposure to dust, smoke, winds and the sun), Vyāyāma (excessive exercise), Karmabhārādhva (excessive hard working, weight lifting and walking long distances), Vegāghāta (suppression of natural urges), Apatarpaṇa (fasting), Āmadoṣa (afflicted with Āma), Abhighāta (injury), Strī (excessive intercourse with women), Kṣayadoṣaprapīḍanaiḥ (afflicted with consumption), Viṣamāśana (irregular dietary habits), Adhyāśana (taking meal before the previous meal has been digested), Anāśana (not taking food at all or very less), Samaśanairapi (simultaneously taking wholesome and unwholesome foods) are the factors producing the disease Hikkā, Śvāsa and Kāsa in the human beings.

Sāmānya Saṃprāpti of Hikkā

- ⦿ मारुतः प्राणवाहीनि स्रोतांस्याविश्य कुप्यति। उरःस्थः कफमुद्धूय हिक्काश्वासान् करोति सः॥
  घोरान् प्राणोपरोधाय प्राणिनां पञ्च पञ्च च ॥ (Ca.Ci.17/17-18)

Due to indulgence in the above mentioned causative factors, the Vāta gets aggravated in the Prāṇavaha srotas, then it disturbs the Kapha located in the chest and produces the disease Hikkā and Śvāsa, each having five types which cause obstruction to the Prāṇa of the person.

- ⦿ प्राणोदकान्नवाहीनि स्रोतांसि सकफोऽनिलः। हिक्काः करोति संरुध्य ...॥ (Ca.Ci.17/21)

Prāṇa Vāta associated with Kapha obstructs the Prāṇa, Anna and Udakavaha srotas and produces the disease Hikkā.

Saṃprāpti Ghaṭaka of Hikkā

Doṣa	Vāta, Kapha
Dūṣya	Rasa
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhya janya Āma
Srotas	Rasa, Prāṇa, Anna, and Udaka vaha
Srotoduṣṭi	Saṅga, Vimārga gamana
Udbhava sthāna	Āmāśaya
Sañcāra sthāna	Prāṇavaha srotas
Vyakta sthāna	Uras

Adhiṣṭhāna	Pitta sthāna
Rogamārga	Abhyantara

Pūrvarūpa of Hikkā

- ⊙ मुखं कषायमरतिर्गौरवं कण्ठवक्षसोः । पूर्वरूपाणि हिक्कानामाटोपो जठरस्य च ॥ (Su.U.50/8)
- ⊙ कण्ठोरसोर्गुरुत्वं च वदनस्य कषायता। हिक्कानां पूर्वरूपाणि कुक्षेराटोप एव च॥ (Ca.Ci.17/19)

Kaṇṭhorasorgurutvaṃ (heaviness in the throat and chest region), Vadanasya kaṣāyatā (astringent taste in the mouth), Kukṣerāṭopa (distension of abdomen) and Arati (excessive uneasiness) are the premonitory symptoms of Hikkā.

Hikkā Bheda and Lakṣaṇās

- ⊙ अन्नजां यमलां क्षुद्रां गम्भीरां महतीं तथा । वायुः कफेनानुगतः पञ्च हिक्काः करोति हि ॥ (Su.U.50/7)
- ⊙ हिध्माः भक्तोद्भवा क्षुद्रा यमला महतीति च। गम्भीरा च .... ॥ (A.H.Ni.4/19)

Hikkā is classified into five types namely, Mahā (Mahatī), Gambhīrā, Vyapetā, Kṣudra and Annajā (Bhaktodbhavā) Hikkā. Instead of Vyapetā Hikkā, Ācārya Suśruta and Vāgbhaṭa described Yamalā Hikkā.

Mahā Hikkā (Mahatī)

⊙ क्षीणमांसबलप्राणतेजसः सकफोऽनिलः। गृहीत्वा सहसा कण्ठमुच्चैर्घोषवतीं भृशम्॥
करोति सततं हिक्कामेकद्वित्रिगुणं तथा। प्राणः स्रोतांसि मर्माणि संरुध्योष्माणमेव च॥
सञ्ज्ञां मुष्णाति गात्राणां स्तम्भं सञ्जनयत्यपि। मार्गं चैवान्नपानानां रुणद्ध्युपहतस्मृतेः॥
साश्रुविप्लुतनेत्रस्य स्तब्धशङ्खच्युतभ्रुवः। सक्तजल्पप्रलापस्य निर्वृतिं नाधिगच्छतः॥
महामूला महावेगा महाशब्दा महाबला। महाहिक्केति सा नृणां सद्यः प्राणहरा मता॥ (Ca.Ci.17/22-26)

In the person having Kṣīṇa māṃsabalaprāṇatejasaḥ (depleted muscle, strength, vital breath and Agni), the Vāta associated with Kapha affects the Kaṇṭha (throat) and produces Uccairghoṣa Hikkā (loud and resonant hiccough sound) characterized with Satataṃ hikkamekadvitriguṇaṃ (one or two or three bouts constantly). The aggravated Vāta and Kapha obstructs the Prāṇavaha srotas, Marmā and Ūṣmāṇa (heat of the body) and produces Saṃjñāṃ muṣṇāti (destroys conciousness), Gatrāṇāṃ sthambhaṃ (stiffness of the body) and Annāpāna mārga ruṇadhi (obstructs the channels of food and drinks). And also it produces Upahata smṛtaḥ (weakens the memory), Sāśruviplutanetrasya (eyes filled with tears), Stabdhaśaṅkha (stiffness in the temples), Cyutabhruvaḥ (eyebrows get displaced), Saktajalpa (adhered or obstructed speech) and Pralāpa (delirium), which does not get relief. This Mahā hikkā is Mahāmūlā (deep rooted), Mahāvegā (forceful paroxysms), Mahāsabdā (loud sound), Mahābalā (extreme severity) and it lead to Sadyaḥ prāṇahara (immediate death) and hence it is named as Mahā hikkā.

⊙ मर्माण्यापीडयन्तीव सततं या प्रवर्तते । देहमायम्य वेगेन घोषयत्यतितृष्यतः ।
महाहिक्केति सा ज्ञेया सर्वगात्रप्रकम्पिनी ॥ (Su.U.50/14)

The hiccup which produces Marmāṇyāpīḍayantīva (crushing pain in the vital parts), Dehamāyamya vegena (stretching out the body forcefully) accompanied with ghoṣa (loud sounds), Ati tṛṣṇa (severe thirst), Sarvagātra kampa (shaking of the whole body) and Satatam yā pravartate (that occurs frequently) is called Mahā hikkā.

Gambhīrā Hikkā

⊙ हिक्कते यः प्रवृद्धस्तु कृशो दीनमना नरः। जर्जरेणोरसा कृच्छ्रं गम्भीरमनुनादयन्॥
सञ्जृम्भन् सङ्क्षिपंश्चैव तथाऽङ्गानि प्रसारयन्। पार्श्वे चोभे समायम्य कूजन् स्तम्भरुगर्दितः॥
नाभेः पक्वाशयाद्वाऽपि हिक्का चास्योपजायते। क्षोभयन्ती भृशं देहं नामयन्तीव ताम्यतः॥
रुणद्ध्युच्छ्वासमार्गं तु प्रनष्टबलचेतसः। गम्भीरा नाम सा तस्य हिक्का प्राणान्तिकी मता॥ (Ca.Ci.17/27-30)

In the person who is Pravṛddha (aged), Kṛśa (emaciated), Dīnamanā (dispirited) and Jarjareṇorasā (feeble chest), the aggravated Vāta along with Kapha produces Hikkā which is characterised by Gambhīramanunādayan (deep and resonant sound) along with Saṃjṛmbha (yawning), Saṃkṣipaṃścaiva tathāṅgāni prasārayan (contraction and extension of the body parts), Pārśve cobhe samāyamya kūjan (while breathing, both the flanks contracts and makes cooing sound) and Stambha rugarditaḥ (stiffness and pain). This hiccup develops from the Nābhi or Pakvāśaya with Kṣobhayanti bhṛśaṃ dehaṃ (agitating the body violently), Dehaṃ Nāmayantīva (bending of the body) and produces Tāmyataḥ (darkness before his eyes). The Hikkā named Gambhīrā hikkā does Ruṇadhdyucchvāsamārga (obstruction of the passage of respiration), and Pranaṣṭabalacetasḥ (destroys the strength and consciousness) which results in death.

⊙ नाभिप्रवृत्ता या हिक्का घोरा गम्भीरनादिनी ॥ शुष्कौष्ठकण्ठजिह्वास्यश्वासपार्श्वरुजाकरी ।
अनेकोपद्रवयुता गम्भीरा नाम सा स्मृता ॥ (Su.U.50/12-13)

Ghora hikkā (violent hiccup) that develops from the Nābhi (umbilical region) and produces Gambhīranādinī (deep sounds) accompanied with Śuṣkauṣṭhakaṇṭhajihvāsya (dryness of the lips, throat, tongue and mouth), Svāsa (dyspnoea), Pārsvarujā (pain in the flanks), Anekopadrava (associated with many complications) is called Gambhīrā hikkā.

Vyapetā Hikkā

⊙ व्यपेता जायते हिक्का याऽन्नपाने चतुर्विधे। आहारपरिणामान्ते भूयश्च लभते बलम्॥
प्रलापवम्यतीसारतृष्णार्तस्य विचेतसः। जृम्भिणो विप्लुताक्षस्य शुष्कास्यस्य विनामिनः॥
पर्याध्मातस्य हिक्का या जत्रुमूलादसन्तता। सा व्यपेतेति विज्ञेया हिक्का प्राणोपरोधिनी॥ (Ca.Ci.17/31-33)

Vyapetā Hikkā manifests after taking the Caturvidha annapāna i.e. eatable, chewable, drinkable and lickable. It gets aggravated after the Āhāra pariṇāma (digestion of food) and produces the symptoms like Pralāpa (delirium), Vamathu (vomiting), Atisāra (diarrhoea).

Tṛṣṇā (thirst), Artasya vicetasḥ (afflicted with absence of mind), Jṛmbhiṇā (yawning), Viplutākṣa (eyes filled with tears), Śuṣkāsya (dryness of mouth), Vināminaḥ (bending of the body) and Paryādhmāta (severe distention of the abdomen). This hiccup which develops from Jatrumūla (root of the neck) and it is Asantatā (bouts of hiccups is short) is known as Vyapetā Hikkā which is Prāṇoparodhinī (obstructs the vital breathe).

Yamalā Hikkā

◉ चिरेण यमलैर्वेगैर्या हिक्का सम्प्रवर्तते ॥ कम्पयन्ती शिरोग्रीवं यमलां तां विनिर्दिशेत् । (Su.U.50/11)

The hiccup which occurs in Yamala vega (double bouts) and at long intervals associated with Śirogrīva kampa (tremors of the head and neck) is called Yamalā Hikkā.

◉ चिरेण यमलैर्वेगैराहारे या प्रवर्तते। परिणामोन्मुखे वृद्धिं परिणामे च गच्छति॥ कम्पयन्ती शिरोग्रीवमाध्मातस्याතितृष्यतः। प्रलापच्छर्द्यतीसारनेत्रविप्लुतिजृम्भिणः॥ यमला वेगिनी हिध्मा परिणामवती च सा। (A.H.Ni.4/23-25)

This hiccup occurs in Yamala vega (double bouts), Cireṇa (at long intervals), Pariṇāmonmukhe (after digestion of food), Vṛddhiṃ pariṇāme ca gacchati (increases during the digestion process) associated with Śirogrīva kampa (tremors of the head and neck), Ādhmāna (distension of abdomen), Ati tṛṣṇa (excessive thirst), Pralāpa (delirium), Chardi (vomiting), Atisāra (diarrhoea), Netravipluta (eyes filled with tears) and Atijṛmbhiṇā (yawning). The hiccup which is related to Pariṇāma (digestion) and occurs in Yamala vega (double bouts) is called Yamalā hikkā or Veginī Hikkā.

Kṣudra Hikkā

◉ क्षुद्रवातो यदा कोष्ठाद्व्यायामपरिघट्टितः। कण्ठे प्रपद्यते हिक्कां तदा क्षुद्रां करोति सः॥
अतिदुःखा न सा चोरःशिरोमर्मप्रवाधिनी। न चोच्छ्वासान्नपानानां मार्गमावृत्य तिष्ठति॥
वृद्धिमायस्यतो याति भुक्तमात्रे च मार्दवम्। यतः प्रवर्तते पूर्वं तत एव निवर्तते॥
हृदयं क्लोम कण्ठं च तालुकं च समाश्रिता। मृद्वी सा क्षुद्रहिक्केति नृणां साध्या प्रकीर्तिता॥ (Ca.Ci.17/34-37)

Due to Vyāyāma (physical exercises), Kṣudravāta (the word implies slightly aggravated or Udāna Vāta) in the Koṣṭha slightly aggravates and reaches the throat, resulting in Kṣudra Hikkā. This type of hiccup does not produce Ati duḥkhā (severe miseries), Na sā coraḥ śiromarmaprabodhinī(doesnotafflictthechest,headandvitalparts),Nacocchvāsānnapānānām mārgamāvṛtya tiṣṭhati (does not obstruct the passage of respiration, food and drinks). Vṛddhimāyasyato yāti bhuktamātre ca mārdavam (it aggravates on exertion and subsides by intake of food in the same way as it started). The doṣas gets located in Hṛdaya, Kloma, Kaṇṭha and Tālukaṃ and produces a mild hiccup; hence it is called as Kṣudra Hikkā, which is said to be curable.

◉ विकृष्टकालैर्या वेगैर्मन्दैः समभिवर्तते ॥ क्षुद्रिका नाम सा हिक्का जत्रुमूलात् प्रधाविता ॥ (Su.U.50/11-12)

The hiccup which occurs with Vegamanda (mild force) at Vikṛṣṭakāla (long intervals) from the Jatrumūla (root of the neck) is known as Kṣudra Hikkā.

Annajā (Bhaktodbhavā) Hikkā

◉ सहसाऽत्यभ्यवहृतैः पानान्नैः पीडितोऽनिलः। ऊर्ध्वं प्रपद्यते कोष्ठान्मद्यैर्वाऽतिमदप्रदैः॥
तथाऽतिरोषभाष्याध्वहास्यभारातिवर्तनैः। वायुः कोष्ठगतो धावन् पानभोज्यप्रपीडितः॥
उरःस्रोतः समाविश्य कुर्याद्धिक्कां ततोऽन्नजाम्। तथा शनैरसम्बन्धं क्षुवंश्चापि स हिक्कते॥
न मर्मबाधाजननी नेन्द्रियाणां प्रबाधिनी। हिक्का पीते तथा भुक्ते शमं याति च साऽन्नजा। (Ca.Ci.17/38-41)

Due to Atyabyavahṛtaiḥpānānnaiḥ (excessive intake of food and drinks), Madya (alcohol) or Atimadapradaiḥ (other intoxicating substances) and Atiroṣabhāṣyādhva hāsyabhārativartanaiḥ (excessive anger, speech, walking long distances, laughing and weight lifting), the Vāyu located in the Koṣṭha gets aggravated and moves in the upward direction; aggaravated Vāyu in Koṣṭha inflicted by food and drinks, moves upward and reaches the Uraḥsrotas (channels in the chest) and produces Annaja Hikkā. This Hikkā occur Śanairsaṃbhandhaṃ kṣuvaṃścāpi (slowly with interruptions along with sneezing), Na marmabādhājananī nendriyāṇāṃ prabādhinī (does not afflicts vital points and sense organs) and gets subsides by Pīte tathā bhukte śamaṃ (intake of food or drinks), is known as Annaja Hikkā.

◉ त्वरमाणस्य चाहारं भुञ्जानस्याथवा घनम् । वायुरन्नैरवस्तीर्णः कटुकैरर्दितो भृशम् ॥
हिक्कयत्यूर्ध्वगो भूत्वा तां विद्यादन्नजां भिषक् ॥ (Su.U.50/9-10)

In the person who takes Tvaraṃmāṇasya cāhāraṃ (food in hurry), Bhuñjānasthāthavā ghanam (who take too much of food and drinks) and Kaṭukairardito bhṛśam (who takes pungent substances regularly), the Vāta gets aggravated and is pushed upwards and produces Hikkā, which is known as Annaja Hikkā

Sādhyāsādhyata of Hikkā

◉ अतिसञ्चितदोषस्य भक्तच्छेदकृशस्य च। व्याधिभिः क्षीणदेहस्य वृद्धस्यातिव्यवायिनः॥
आसां या सा समुत्पन्ना हिक्का हन्त्याशु जीवितम्। यमिका च प्रलापार्तितृष्णामोहसमन्विता॥
अक्षीणश्चाप्यदीनश्च स्थिरधात्विन्द्रियश्च यः। तस्य साधयितुं शक्या यमिका हन्त्यतोऽन्यथा॥ (Ca.Ci.17/42-44)

The Hikkā manifested in a person having Atisañcita doṣa (accumulation of doṣas), Bhaktaccheda kṛśa (become emaciated due to discontinuation of food), Vyādhibhiḥ Kṣīṇa dehasya (who become emaciated by the diseases), Vṛddha (old aged) and Ativyavāyinaḥ (indulging in excessive sexual interourse) should be rejected as it takes his life very quickly.

The Yamikā hikkā associated with Pralāpa (delirium), Arti (pain), Tṛṣṇā (thirst) and Mohā (unconsciousness) is incurable whereas it is curable in an individual who is Akṣīṇā (not emaciated), Apyadīna (not indigent) and Sthiradhātvendriya (has stable dhātus and sense organs). Otherwise it is fatal.

◉ आयम्यते हिक्काऽतोङ्गानि यस्य दृष्टिश्चोर्ध्वं ताम्यते यस्य गाढम् ।
क्षीणोऽन्नद्विट् कासते यश्च हिक्की तौ द्वावन्त्यौ वर्जयेद्धिक्कमानौ ॥ (Su.U.50/15)

When during a bout of hiccup, Āyamyate angāni (the body becomes stretched), Dṛṣṭiścordhvaṃ (eyes moves upwards), Tāmyate yasya gāḍam (severe darkness appears all around) and as the one suffering from Kṣīṇa (weakness), Annadviṭ (aversion to food and Kāsate (cough) and also one who is suffering from last two types of Hikkā (Gambhīrā and Mahatī Hikkā) should be given up as it is incurable.

- ....तासु साधयेत्। आद्ये द्वे, वर्जयेदन्त्ये सर्वलिङ्गां च वेगिनीम्॥
  सर्वाश्च सञ्चितामस्य स्थविरस्य व्यवायिनः। व्याधिभिः क्षीणदेहस्य भक्तच्छेदक्षतस्य वा। (A.H.Ni.4/29-30)

Among these, the first two types of Hikkā (Annaja and Kṣudra) should be treated; the last two types of Hikkā (Mahā and Gambhīrā) should be rejected and also in the person who is suffering from Yamalā (Veginī) Hikkā with presence of all of its features should be rejected. In persons who have Sañcitāma (accumulated Āma) in the body, Sthavirasya (who become old aged), Vyavāyinaḥ (indulging in excessive sexual interourse), Vyādhibhiḥ Kṣīṇadehasya bhaktacchedakṣatasya (who become emaciated by the diseases and discontinuation of food) should be rejected.

In Contemporary science,

Hikkā can be correlated with Singultus (hiccup) in contemporary science. Hiccups are characterised by the sudden spasmodic contraction of the diaphragm followed by sudden closure of the glottis, which produces a characteristic sound of air being expelled violently from the lungs and it may be classified in to transient hiccup (lasting for seconds or minutes), persistent hiccups (lasting more than 48 hours) and intractable hiccups (lasting more than a month or two). Hiccup is caused by various factors such as

- Irritation of the phrenic or vagus nerve by gatro-oesophageal reflux, laryngitis, hair or foreign-body irritation of tympanic membrane, goitre and tumour or cyst in the neck,
- Central nervous disorders like CVA, encephalitis, meningitis, brain abscess, head trauma, brain tumours, syringomyelia and hydrocephalus etc.
- GI disorders like gastritis, oesophagitis, pancreatitis, peptic ulcer disease, CA of stomach and pancreas, abdominal abscesses, gallbladder disease, inflammatory bowel disease, hepatitis, aerophagia and bowel obstruction
- Cardiovascular disease like Myocardial infarction, pericarditis
- Toxic states due to alcoholism, diabetes mellitus, uraemia, hypocalcaemia and hyponatraemia etc.
- Psychogenic causes such as anorexia nervosa, stress, schizophrenia, etc.

MCQ's

1. Hidhmā is the synonym of

a) Hikkā
b) Śvāsa
c) Kāsa
d) Rājayakṣmā

2. Doṣa predominance in Hikkā

a) Vātapitta
b) Vātakapha
c) Pittakapha
d) Sannipātaja

3. Hikkā is of .... types

a) 4
b) 5
c) 6
d) 7

4. According to Caraka, the type of Hikkā not mentioned is

a) Mahat
b) Gambhīrā
c) Yamala
d) Vyapetā

5. Pūrvarūpa of Hikkā includes

a) Amlāsyata
b) Tiktāsyata
c) Kaṣāyāsyata
d) Madhurāsyata

6. सर्वगात्रप्रकम्पिनी is the feature of

a) Mahā Hikkā
b) Gambhīrā Hikkā
c) Yamala Hikkā
d) Vyapetā Hikkā

7. नाभेः पक्वाशयाद्वाऽपि हिक्का चास्योपजायते is the feature of

a) Mahā Hikkā
b) Gambhīrā Hikkā
c) Yamala Hikkā
d) Vyapetā Hikkā

8. Which type of Hikkā originates after the intake of Caturvidha Annapāna

a) Mahā Hikkā
b) Gambhīrā Hikkā
c) Yamala Hikkā
d) Vyapetā Hikkā

9. Veginī Hikkā is also known as
   a) Mahā Hikkā b) Gambhīrā Hikkā
   c) Yamala Hikkā d) Kṣudra Hikkā
10. Which type of Hikkā originates from Jatrumūla
   a) Kṣudra Hikkā b) Mahā Hikkā
   c) Gambhīrā Hikkā d) Yamala Hikkā

Answer Keys

1	a	2	b	3	b	4	c	5	c
6	a	7	b	8	d	9	c	10	a

Short Answer Questions

1. Explain Nidāna and Saṃprāpti of Hikkā
2. Explain the Bheda and lakṣaṇās of Hikkā
3. Explain Sādhyāsādhyata of Hikkā

◆

TOPIC 13

Śvāsa

Introduction

Śvāsa encompasses a physiological process vital to life, yet it can also manifest as a disorder associated with the Prāṇavaha srotas. This condition may present independently or as a symptom of other disorders. Like Hikkā, Śvāsa is also characterized by a disturbance in the Kapha and Vata doṣas, originating from the site of Pitta, leading to the depletion of the Rasādi dhātu located in the Hṛdaya (heart). Consequently, treating this condition poses notable challenges. Similar to Hikkā, Śvāsa may result in Prāṇānāśu nikuntataḥ (immediate death), often observed in the terminal phases of life, irrespective of the coexistence of other pathological conditions. Without proper intervention, Śvāsa can prove as harmful to the patient as Āśīviṣa (snake poison).

Nirūkti and Paribhāṣā of Śvāsa

- ⦿ श्वसित्यनेनेति श्वासः॥ (SKD)
- ⦿ श्वसितौति श्वासः॥ (SKD)

That which produces 'sigh' is called Śvāsa

- ⦿ श्वास इति वेगवदूर्ध्ववातत्वं॥ (Āḍ.Śā.P.7/245)

The disease which originates from the forceful upward movement of Vāta is called Śvāsa

- ⦿ विहाय प्रकृतिं वायुः प्राणोऽथ कफसंयुतः । श्वासयत्यूर्ध्वगो भूत्वा तं श्वासं परिचक्षते ॥ (Su.U.51/4)
- ⦿ प्राणो वायुः, प्रकृतिं विहाय विगुणो भूत्वेत्यर्थः, ऊर्ध्वगो भूत्वा तथा कफसंयुतः सन् यदा श्वासयति तं बुधाः श्वासं परिचक्षते। (Ḍa.Su.U.51/4)

The aggravated Prāṇa Vāta combines with Kapha moves Ūrdhva Gati (upwards) and produces gasping and labored breathing which is called Śvāsa.

Śvāsa Nidāna

- ⊙ रजसाधूमवाताभ्यांशीतस्थानाम्बुसेवनात्।व्यायामाद्ग्राम्यधर्माध्वरूक्षान्नविषमाशनात्॥
  आमप्रदोषादानाहाद्रौक्ष्यादत्यपतर्पणात्। दौर्बल्यान्मर्मणो घाताद्द्वन्द्वाच्छुद्ध्यतियोगतः॥
  अतीसारज्वरच्छर्दिप्रतिश्यायक्षतक्षयात्। रक्तपित्तादुदावर्ताद्विसूच्यलसकादपि॥
  पाण्डुरोगाद्विषाच्चैव प्रवर्तेते गदाविमौ। निष्पावमाषपिण्याकतिलतैलनिषेवणात्॥
  पिष्टशालूकविष्टम्भि विदाहिगुरुभोजनात्। जलजानूपपिशितदध्यामक्षीरसेवनात्॥
  अभिष्यन्द्युपचाराच्च श्लेष्मलानां च सेवनात्। कण्ठोरसः प्रतीघाताद्विबन्धैश्च पृथग्विधैः॥ (Ca.Ci.17/11-16)

Rajodhūmavātābhyāṃ (exposure to dust, smoke and winds), Śītasthānāmbusevanāt (residing in cold environment and intake of cold water), Vyāyāma (excessive exercise), Grāmyadharmā (excessive sexual intercourse), Adhva (walking long distances), Rūkṣānna (intake of non-unctuous food), Viṣamāśana (irregular dietary habits), Āmapradoṣā (afflicted with Āma), Ānāha (abdominal distention and constipation),Raukṣyāt (non- unctuousness), Apatarpaṇa (excessive fasting), Daurbalya (weakness), Marmaṇoghāta (injury to the vital parts), Dvandvā (use of cold and hot substances or wholesome and unwholesome substances), Śuddhāyatiyogataḥ (excessive purificatory procedures) and also due to complications of diseases such as Atisāra, Jvara, Chardi, Pratiśyāya, Kṣata, Kṣaya, Raktapitta, Udāvarta, Visūcī, Alasaka, Pāṇḍuroga ad Viṣa. And also, the disease Hikkā and Śvāsa get manifested due to intake of Niṣpāva, Māṣa, Pinyāka, Tilataila, Piṣṭaśālūka (pastry and stem of lotus), Viṣṭambhividāhigurubhojanāt (intake of food substances which are constipated, producing burning sensation and heavy), Jalajānūpapiśitadadhyāmakṣīrasevanāt (intake of meat of aquatic and marshy animals, curds and unboiled milk), Abhiṣyandi (channels blocking substances), Śleṣmalānāṃ ca sevanat (use of Kapha aggravating factors); Kaṇṭhorasaḥ pratighāta (injury to throat and chest) and Vibandhaśca pṛthagvidaiḥ (various types of constipation)

- ⊙ विदाहिगुरुविष्टम्भिरूक्षाभिष्यन्दिभोजनैः। शीतपानाशनस्थानरजोधूमातपानिलैः॥
  व्यायामकर्म भाराध्ववेगाघातापतर्पणैः । हिक्का श्वासश्च कासश्च नृणां समुपजायते ॥ (M.N.12/1-2)
- ⊙ विदाहिगुरुविष्टम्भिरूक्षाभिष्यन्दिभोजनैः। शीतपानाशनस्थानरजोधूमानिलानलैः ॥
  व्यायामकर्म भाराध्ववेगाघातापतर्पणैः । आमदोषाभिघातस्त्रीक्षयदोषप्रपीडनैः ॥
  विषमाशनाध्यनशनैस्तथा समशनैरपि । हिक्का श्वासश्च कासश्च नृणां समुपजायते ॥ (Su.U.50/3-5)

Indulging in food which are Vidāhi (producing burning sensation), Guru (heavy), Viṣṭambhi (constipation), Rūkṣa (non-unctuous) and Abhiṣyandi bhojanaḥ (channels blocking substances), Śītapānāsanasthāna (intake of cold water and exposure to cold environment), Rajodhūmānillānalaiḥ (exposure to dust, smoke, winds and the sun), Vyāyāma (excessive exercise), Karmabhārādhva (excessive hard working, weight lifting and walking long distances), Vegāghāta (suppression of natural urges), Apatarpaṇa (fasting), Āmadoṣā (afflicted with Āma), Abhighāta (injury), Strī (excessive intercourse with women), Kṣayadoṣaprapīḍanaiḥ (afflicted with consumption), Viṣamāśana (irregular dietary habits), Adhyāśana (taking meal before the previous meal has been digested), Anāśana (not taking food at all or very less), Samaśanairapi (simultaneously taking wholesome and unwholesome foods) are the factors producing the disease Hikkā, Śvāsa and Kāsa in the human beings.

- ⊙ कासवृद्ध्या भवेच्छ्वासः पूर्वैर्वा दोषकोपनैः। आमातिसारवमथुविषपाण्डुज्वरैरपि॥
  रजोधूमानिलैर्मर्मघातादतिहिमाम्बुना। (A.H.Ni.4/1-2)

Śvāsa is caused by Kāsavṛddhi (increase of cough) or Pūrva doṣakopanaiḥ (aggravation of doṣas described earlier) and also by the diseases Amātisāra, Vamathu, Viṣa, Pāṇḍu and Jvara; and also Rajodhūmānila (exposure to dust, smoke and wind), Marmaghātat (injury to vital points) and Himāmbuna (drinking of very cold water).

Sāmānya Saṃprāpti of Śvāsa

- ⊙ मारुतः प्राणवाहीनि स्रोतांस्याविश्य कुप्यति। उरःस्थः कफमुद्धूय हिक्काश्वासान् करोति सः॥
  घोरान् प्राणोपरोधाय प्राणिनां पञ्च पञ्च च ॥ (Ca.Ci.17/17-18)

Due to indulgence in the above mentioned causative factors, the Vāta gets aggravated in the Prāṇavaha srotas, then it disturbs the Kapha located in the chest and produces the disease Hikkā and Śvāsa, each having five types which cause obstruction to the Prāṇa of the person.

- ⊙ यदा स्रोतांसि संरुध्य मारुतः कफपूर्वकः। विष्वग्व्रजति संरुद्धस्तदा श्वासान्करोति सः॥ (Ca.Ci.17/45)

Vāta preceded by Kapha obstructs the Prāṇa, Udaka and Annavaha srotas and itself being obstructed by Kapha moves to all places and produces the disease Śvāsa.

Saṃprāpti Ghaṭaka of Śvāsa

Doṣa	Vāta, Kapha
Dūṣya	Rasa
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhyajanya Āma
Srotas	Rasa, Prāṇa, Anna and Udaka vaha
Srotoduṣṭi	Saṅga, Vimārga gamana
Udbhava sthāna	Āmāśaya
Sañcāra sthāna	Prāṇavaha srotas
Vyakta sthāna	Uras

Adhiṣṭhāna	Pitta sthāna
Rogamārga	Abhyantara

Pūrvarūpa of Śvāsa

- ⊙ आनाहः पार्श्वशूलं च पीडनं हृदयस्य च। प्राणस्य च विलोमत्वं श्वासानां पूर्वलक्षणम्॥ (Ca.Ci.17/20)
- ⊙ प्राग्रूपं तस्य हृत्पीडा भक्तद्वेषोऽरतिः परा । आनाहः पार्श्वयोः शूलं वैरस्यं वदनस्य च ॥ (Su.U.51/6)

Hṛtpārśva śūla (pain in the cardiac region and flanks), Bhaktadveśa (aversion to food), Arati (excessive uneasiness), Ānāha (constipation with abdominal distention), Prāṇasya vilomata (upward movement of Prāṇa Vāta) and Vadana vairasya (bad taste in the mouth) are the premonitory symptoms of Śvāsa.

Śvāsa Bheda and Lakṣaṇās

- ⊙ क्षुद्रकस्तमकश्छिन्नो महानूर्ध्वश्च पञ्चधा । भिद्यते स महाव्याधिः श्वास एको विशेषतः । (Su.U.51/5)

Śvāsa - a Māhavyādhi is one only but is classified into five types namely, Kṣudra, Tamaka, Chinna, Mahān and Ūrdhva Śvāsa

- ⊙ क्षुद्रको वातिकः श्लेष्मभूयिष्ठस्तमकः स्मृतः। छिन्नः पित्तप्रधानः स्यादन्यौ मारुतकोपजौ"- इति। (Ḍa.Su.U.51/5)
- ⊙ वाताधिको भवेत् क्षुद्रस्तमकस्तु कफोद्भवः । कफवाताधिकश्चैव संसृष्टश्छिन्नसञ्ज्ञकः ।
  श्वासो मारुतसंसृष्टो महानूर्ध्वस्तथा(तो) मतः ॥ (M.N.12/15)

Ācārya Ḍalhaṇa described the doṣa predominance in the five types of Śvāsa. Kṣudra Śvāsa, Mahān, and Ūrdhva Śvāsa are Vāta predominant, whereas Tamaka Śvāsa is Kapha predominant and Chinna Śvāsa is Pitta predominant. Ācārya Mādhavakara mentioned that the Chinna Śvāsa is Kapha and Vāta predominant.

Śvāsa Bheda	Doṣa Pradhāna	
	Suśruta	Mādhavakara
Kṣudra Śvāsa	Vāta Pradhāna	Vāta Pradhāna
Mahān Śvāsa	Vāta Pradhāna	Vāta Pradhāna
Ūrdhva Śvāsa	Vāta Pradhāna	Vāta Pradhāna
Tamaka Śvāsa	Kapha Pradhāna	Kapha Pradhāna
Chinna Śvāsa	Pitta Pradhāna	Kapha and Vāta Pradhāna

Kṣudra Śvāsa

- ⊙ रूक्षायासोद्भवः कोष्ठे क्षुद्रो वात उदीरयन्। क्षुद्रश्वासो न सोऽत्यर्थं दुःखेनाङ्गप्रबाधकः॥
  हिनस्ति न स गात्राणि न च दुःखो यथेतरे। न च भोजनपानानां निरुणद्ध्युचितां गतिम्॥

नेन्द्रियाणां व्यथां नापि काञ्चिदापादयेद्रुजम्। स साध्य उक्तो बलिनः सर्वे चाव्यक्तलक्षणाः॥ (Ca.Ci.17/65-66)

Due to indulgence of Rūkṣa (non-unctuousness diet and activity) and Āyāsā (exertion), Vāta is mildly vitiated in Koṣṭha (abdomen) and moves upwards direction and produces Kṣudra Śvāsa, which does afflicts the various functions of the body parts and also does not harm or distress the body like other types of Śvāsa. It neither obstructs the passage of food and drinks nor produces any pain to the sense organs. It is said to be Sādhya (curable) in the strong patient with incomplete manifestation of the symptoms.

- ⊙ किञ्चिदारभमाणस्य यस्य श्वासः प्रवर्तते । निषण्णस्यैति शान्तिं च स क्षुद्र इति सञ्ज्ञितः ॥ (Sū.U.51/7)

In Kṣudra Śvāsa, the patient develops Śvāsa (breathlessness) on slight exertion and it gets relieved in sitting posture.

Tamaka Śvāsa

- ⊙ तमसेति अन्धकारेण, किंवा तमसा तमोगुणेन। (Ca.Ca.Ci.17/64)

The term 'Tama' means 'Andhakāraṇa' which means darkness or 'Tama' means 'Tamoguna'. During an acute attack of Śvāsa, the patient feels as if entering in darkness. Hence it may be named as Tamaka Śvāsa.

- ⊙ प्रतिलोमं यदा वायुः स्रोतांसि प्रतिपद्यते। ग्रीवां शिरश्च सङ्गृह्य श्लेष्माणं समुदीर्य च॥
  करोति पीनसं तेन रुद्धो घुर्घुरुकं तथा। अतीव तीव्रवेगं च श्वासं प्राणप्रपीडकम्॥
  प्रताम्यत्यतिवेगाच्च कासते सन्निरुध्यते। प्रमोहं कासमानश्च स गच्छति मुहुर्मुहुः॥
  श्लेष्मण्यमुच्यमाने तु भृशं भवति दुःखितः। तस्यैव च विमोक्षान्ते मुहूर्तं लभते सुखम्॥
  अथास्योद्ध्वंसते कण्ठः कृच्छ्राच्छक्नोति भाषितुम्। न चापि निद्रां लभते शयानः श्वासपीडितः॥
  पार्श्वे तस्यावगृह्णाति शयानस्य समीरणः। आसीनो लभते सौख्यमुष्णं चैवाभिनन्दति॥
  उच्छ्रिताक्षो ललाटेन स्विद्यता भृशमर्तिमान्। विशुष्कास्यो मुहुः श्वासो मुहुश्चैवावधम्यते॥
  मेघाम्बुशीतप्राग्वातैः श्लेष्मलैश्चाभिवर्धते। स याप्यस्तमकश्वासः साध्यो वा स्यान्नवोत्थितः॥ (Ca.Ci.17/55-62)

Vāta moves upward in the Prāṇavaha Srotas and get into Grīvā (neck) and Śiras (head), aggravates the Kapha and produces Pīnasa (coryza) which cause obstruction and produces Ghurghuraka (whistling sound) and Atīva tīvravega śvāsa (extremely severe paroxysm of respiration) which afflicts the Prāṇa. During this severe attack, the patient gets Ativega Kāsate sannirudhyate (coughs with forceful paroxysm and becomes motionless), Pramoha kāsamānaśca sa gacchati muhurmuhuḥ (he faints repeatedly while coughing). Śleṣmaṇyamucyamāne tu bṛśaṃ bhavati duḥkhitaḥ (he becomes distressed due to absence of expectoration), Tasyaiva ca vimokṣānte muhūrtaṃ labhate sukham (gets relieved after expectoration), Athāsyoddhvaṃsate kaṇṭhaḥ kṛcchrācchaknoti bhāṣitum (due to hoarseness of voice, he speaks with difficulty), Na cāpi nidrāṃ labhate śayanaḥ śvāsapīḍitaḥ (he does not get proper sleep because śvāsa aggravates in lying position), Pārśve tasyāvagṛhṇāti śayanasya samīraṇaḥ (in lying position, the śvāsa aggravates because the aggravated Vāta afflicts his flanks), Āsīno labhate saukhyamuṣṇaṃ caivābhinandati (the patients feels comfortable in sitting posture and by hot things), Ucchritākṣau (widely opened eyes), Lalāṭena sviddyatā (sweating in his forehead), Bhṛśamartimān (extremely distressed), Viśuṣkāsyo muhuḥ (dryness of the mouth), Śvāso muhuścaivāvadhamyate (his body trembles during an attack of Śvāsa), Meghāmbuśītaprāgvātaiḥ śleṣmaiścābhivardhate (condition aggravates during cloudy or rainy or cold weather, easterly wind and indulges in Kapha increasing diet and activites) and it is Yāpya (palliable), however recently manifested is Sādhya (curable).

◉ ज्वरमूर्च्छापरीतस्य विद्यात् प्रतमकं तु तम्। उदावर्तरजोऽजीर्णक्लिन्नकायनिरोधजः॥
तमसा वर्धतेऽत्यर्थं शीतैश्चाशु प्रशाम्यति। मज्जतस्तमसीवाऽस्य विद्यात् सन्तमकं तु तम्॥ (Ca.Ci.17/63-64)

If Tamaka Śvāsa is associated with Jvara (fever) and Mūrcchā (fainting), then it is called Pratamaka Śvāsa.

The Pratamaka Śvāsa is known as Santamaka when it is caused by Udāvarta (upward movement of Vāta), Rajo (dust), Ajīrṇa (indigestion), Klinnakāya (humidity) and Nirodhaja (suppression of natural urges), as it results in increase of Tamoguna (darkness) and the patient feels as if sinking into deep darkness. This condition gets pacified by cold regimens.

◉ तृट्स्वेदवमथुप्रायः कण्ठघुर्घुरिकान्वितः । विशेषाद्दुर्दिने ताम्येच्छ्वासः स तमको मतः ॥
घोषेण महताऽऽविष्टः सकासः सकफो नरः । यः श्वसित्यबलोऽन्नद्विट् सुप्तस्तमकपीडितः ॥
स शाम्यति कफे हीने स्वपतश्च विवर्धते । मूर्च्छाज्वराभिभूतस्य ज्ञेयः प्रतमकस्तु सः ॥ (Sū.U.51/8-10)

Śvāsa (breathlessness) associated with Tṛṭ (thirst), Sveda (perspiration), Vamathu (vomiting) and Kaṇṭhaghurghurikā (whistling sound in the throat) and it gets aggravated specially in Durdina (bad or rainy or cloudy weather) is called Tamaka Śvāsa. In this, the patient produces loud sounds with cough and expectoration. Due to this, the patient become weak and develops aversion to food.

The attack of Śvāsa (breathlessness) is more common during sleep and it gets relieved after expectorating of phlegm and gets aggravated on lying down. If the patient of Tamaka Śvāsa develops Mūrcchā (fainting) and Jvara (fever), then it is called Pratamaka Śvāsa.

Chinna Śvāsa

◉ यस्तु श्वसिति विच्छिन्नं सर्वप्राणेन पीडितः। न वा श्वसिति दुःखार्तो मर्मच्छेदरुगर्दितः॥
आनाहस्वेदमूर्च्छार्तो दह्यमानेन वस्तिना। विप्लुताक्षः परिक्षीणः श्वसन् रक्तैकलोचनः॥
विचेताः परिशुष्कास्यो विवर्णः प्रलपन्नरः। छिन्नश्वासेन विच्छिन्नः स शीघ्रं प्रजहात्यसून्॥ (Ca.Ci.17/52-54)

Due to obstruction in the entire Prāṇavaha srotas, the person is afflicted with Vicchinnaṃ śvāsa (interruption or stoppage of breathing) and he suffers from Duḥkhārto marmacchedarugarditaḥ (intense pain as if cutting of the vital organs), Ānāha (constipation with abdominal distension), Sveda (perspiration), Mūrcchā (fainting), Dahyamānena Bastina (burning sensation in the bladder), Viplutākṣaḥ (tears in the eyes), Parikṣīṇaḥ (emaciation of the body), Śvasan raktaivalocanaḥ (his one eye become red like blood while struggling for breathing), Vicetāḥ (disorientation of mind), Pariśuṣkāsyo (dryness of mouth), Vivarṇa (discoloration of skin), Pralāpa (delirium) and Vicchinnaḥ (vimukta sandhi bandhaḥ - looseness of joints) are the signs and symptoms of Chinna Śvāsa. The person suffering from this condition dies quickly.

◉ आध्मातो दह्यमानेन बस्तिना सरुजं नरः । सर्वप्राणेन विच्छिन्नं श्वस्याच्छिन्नं तमादिशेत् । (Sū.U.51/11)

In Chinna Śvāsa, the patient develops Vicchinnaṃ śvāsa (intermittent breathing pattern) and it is associated with Ādhmāto (distenstion of abdomen) and Dahyamānena bastinā sarujaṃ (burning sensation in the bladder with pain).

Mahā Śvāsa

◉ उद्धूयमानवातो यः शब्दवदुःखितो नरः। उच्चैः श्वसिति संरुद्धो मत्तर्षभ इवानिशम्॥
प्रनष्टज्ञानविज्ञानस्तथा विभ्रान्तलोचनः। विकृताक्ष्याननो बद्धमूत्रवर्चा विशीर्णवाक्॥
दीनः प्रश्वसितं चास्य दूराद्विज्ञायते भृशम्। महाश्वासोपसृष्टः स क्षिप्रमेव विपद्यते॥ (Ca.Ci.17/46-48)

In Duḥkhito naraḥ (distressed person), the aggravated Vāta moves upwards and produces Śabdavat ucchaiḥ śvāsa (long deep breath with sound) constantly like Mattarṣabha (intoxicated bull) due to obstruction in the Prāṇavaha srotas associated with Pranaṣṭa Jñāna Vijñāna (loses all his knowledge and senses), Vibhrānta locana (confused look), Vikṛtākṣhyānano (his eyes and face become abnormal), Baddhamūtravarcā (retention of urine and faeces), Viśīrṇavāk (broken voice), Dīnaḥ praśvasitaṃ (his breathing sounds heard from long distance) are the clinical features of Mahā Śvāsa, which kills the person quickly.

◉ निःसञ्ज्ञः पार्श्वशूलार्तः शुष्ककण्ठोऽतिघोषवान् । संरब्धनेत्रस्त्वायम्य यः श्वस्यात् स महान् स्मृतः (Sū.U.51/12)

In Mahān Śvāsa, the patient becomes Niḥsaṃjñaḥ (unconscious) associated with Pārśvaśūla (pain in the flanks), Śuṣkakaṇṭha (dryness of the throat), Atighoṣa (loud sound), Saṃrabda netra (fixed gaze or stare) and Āyamya yaḥ śvasyāt (breathes with stretched body).

Ūrdhva Śvāsa

◉ . दीर्घं श्वसिति यस्तूर्ध्वं न च प्रत्याहरत्यधः। श्लेष्मावृतमुखस्रोताः क्रुद्धगन्धवहार्दितः॥
ऊर्ध्वदृष्टिर्विपश्यंश्च विभ्रान्ताक्ष इतस्ततः। प्रमुह्यन् वेदनार्तश्च शुष्कास्योऽरतिपीडितः॥
ऊर्ध्वश्वासे प्रकुपिते ह्यधःश्वासो निरुध्यते। मुह्यतस्ताम्यतश्चोर्ध्वं श्वासस्तस्यैव हन्त्यसून्॥ (Ca.Ci.17/49-51)

In Ūrdhva Śvāsa, the patient develops Dīrghaṃ ūrdhva śvasiti (prolonged expiration) and Na ca Pratyāharatyadhaḥ (inability of or does not take inspiration) due to obstruction of Mukha (mouth) and Srotas by the aggravated Kapha which leads to aggravation of Vāta and it is associated with Ūrdhva dṛṣṭhi (looks upwards), Vipaśya (sees here and there), Vibhrāntākṣa (confused look), Pramoha (delusion), Vedana (pain), Śuṣkāsyo (dryness of mouth), Aratipīḍitaḥ (seized with distress). Ūrdhvaśvāse prakupite hyadhaḥ śvāso nirudhyate (due to increased expiration and is unable to bring it down or inspiration is obstructed) which leads to Moha (delusion) and Tama (darkness before eyes). Ūrdhva Śvāsa kills the patient in short period.

- ⦿ मर्मस्वायम्यमानेषु श्वसन्मूढो मुहुश्च यः । ऊर्ध्वप्रेक्षी हतरवस्तमूर्ध्वश्वासमादिशेत् ॥ (Sū.U.51/13)

In Ūrdhva Śvāsa, the patient develops Marmasvāyamyamāneṣu śvasan muhuśca (stretching type of pain in the vital areas with each stroke of breath repeatedly) along with Mūḍho (confused state), Ūrdhvaprekṣī (upturned eyes) and Hatasvara (defective voice).

Differentiate Pañca Śvāsa

	Kṣudra	Tamaka	Chinna	Mahā	Ūrdhva
Doṣa	Vāta	Kapha	Pitta	Vāta	Vāta
Śvāsa	Rūkṣāy-asottha śvāsa	Atīva tīvravega śvāsa	Vicchinnaṃ śvāsa	Ucchaiḥ śvāsa	Dīrghaṃ ūrdhva śvasiti
Śabda	–	Ghurghuraka	–	Mattarṣabha	–
Cetas	–	Pramoha, Tama	Vicetāḥ, Mūrcchā	Pranaṣṭa Jñāna Vijñāna	Pramoha, Tama
Netra	–	Ucchritākṣau	Viplutākṣaḥ, Raktaivalocanaḥ	Vibhrānta locana, Vikṛtākṣhyānana	Ūrdhva dṛṣṭhi, Vibhrāntākṣa
Śūla	–	Pārśve tasyāvagṛhṇāti śayanasya samīraṇaḥ	Duḥkhārto marmaccheda rugarditaḥ	Pārśvaśūla	Marmasvāyamya māneṣu śvasan muhuśca
Vāk	–	Athāsyoddhva-ṃsate kaṇṭhaḥ kṛcchrācchaknoti bhāṣitum	Pralāpa	Viśīrṇavāk	Hatasvara
Āsya		Viśuṣkāsya	Pariśuṣkāsya	–	Śuṣkāsya
Sādhyā-sādhyata	Sādhya	Yāpya	Asādhya	Asādhya	Asādhya

Sādhyāsādhyata of Śvāsa

- ⦿ क्षुद्रः साध्यतमस्तेषां तमकः कृच्छ्र उच्यते । त्रयः श्वासा न सिध्यन्ति तमको दुर्बलस्य च ॥ (Sū.U.51/14)

Kṣudra Śvāsa is Sādhya (curable), Tamaka Śvāsa is Kṛcchra Sādhya (curable with difficulty), whereas the other three varieties of Śvāsas like Chinna, Mahān and Ūrdhva Śvāsa and Tamaka Śvāsa in Durbala (weak patient) is Asādhya (incurable).

- ⦿ एषां प्राणहरा वर्ज्या घोरास्ते ह्याशुकारिणः॥ भेषजैः साध्ययाप्यांस्तु क्षिप्रं भिषगुपाचरेत्।
  उपेक्षिता दहेयुर्हि शुष्कं कक्षमिवानलः॥ (Ca.Ci.17/68-69)

Among these (Hikkā and Śvāsa), those already described as fatal i.e. Mahā (Mahatī) Hikkā, Gambhīrā Hikkā, Vyapetā Hikkā, Mahā Śvāsa and Ūrdhva Śvāsa should not be treated. While the curable and palliable types of Hikkā and Śvāsa should be treated immediately because if neglected kills the patient in the same way as the fire burns the dry tree.

In Contemporary science,

Among the five types of Śvāsa, Mahā śvāsa, Ūrdhva śvāsa and Chinna śvāsa are seen in the end stage of various diseases such as diabetic ketoacidosis, cardiac failure, renal failure, brain injury or brain tumour, acute cardiogenic pulmonary oedema, etc. Kṣudra Śvāsa is seen as a symptom in many diseases than a disease itself and Tamaka Śvāsa can be correlated to bronchial asthma in contemporary science.

Śvāsa Bheda	Pratyātmaka Lakṣaṇa	Modern Parlance
Mahā śvāsa	Śabdavat ucchaiḥ śvāsa saṣruddho mattarṣabha (long deep breath associated with loud sound continuously like intoxicated bull)	Kussmaul's respiration is characterized by deep, sighing, rapid breathing at a steady rate and with a hissing sound, indicative of hyperpnoea with increased rate and depth of respiration
Ūrdhva śvāsa	Dīrghaṃ ūrdhva śvasiti (prolonged expiration) and Na ca Pratyāharatyadhaḥ (inability of or does not take inspiration) due to obstruction of Mukha (mouth) and Srotas by the aggravated Kapha	COPD is characterized by both shortness of breath during physical activity and prolonged expiration due to airflow obstruction
Chinna śvāsa	Vicchinnaṃ śvāsa (interruption or stoppage of breathing)	Cheyne-Stokes respiration (CSR) is characterized by a cyclical breathing pattern involving a sequence of apnea, followed by a gradual increase in respiratory frequency and tidal volume, and then a subsequent decline in both parameters until the onset of the next apneic period.
Kṣudra Śvāsa	Ārabhamāṇa śvāsa pravartate niṣaṇṇasyaiti śāntiṃ (breathlessness on slight exertion and it gets relieved in sitting posture).	Dyspnoea on exertion refers to a condition wherein an individual experiences shortness of breath or difficulty breathing while engaged in physical activity.
Tamaka Śvāsa	Ghurghuraka (whistling sound) and Atīva tīvravega śvāsa (extremely severe paroxysm of respiration) which afflicts the Prāṇa.	Bronchial Asthma is characterized by episodes of wheezing, coughing, chest tightness and Shortness of breath.

MCQ's

1. Doṣa Predominance in Śvāsa

a) Vātakapha
b) Vātapitta
c) Kaphapitta
d) Sannipātaja

2. Śvāsa is of .... types

a) 2
b) 4
c) 5
d) 6

3. आसीनो लभते सौख्यमुष्णं चैवाभिनन्दति is mentioned under

a) Chinna Śvāsa
b) Mahā Śvāsa
c) Tamaka Śvāsa
d) None of the above

4. According to Mādhavakara, Doṣa Predominance in Tamaka Śvāsa

a) Vāta Pradhāna
b) Pitta Pradhāna
c) Kapha Pradhāna
d) Kaphavāta Pradhāna

5. Pratamaka Śvāsa is associated with which of the following conditions

a) Jvara and Kāsa
b) Jvara and Mada
c) Jvara and Mūrcchā
d) Jvara and Hikkā

6. According to Suśruta, Doṣa Predominance in Chinna Śvāsa

a) Vāta Pradhāna
b) Pitta Pradhāna
c) Kapha Pradhāna
d) Kaphavāta Pradhāna

7. रक्तैकलोचनः is the feature of

a) Chinna Śvāsa
b) Mahā Śvāsa
c) Tamaka Śvāsa
d) None of the above

8. विभ्रान्तलोचनः is the feature of

a) Chinna Śvāsa
b) Mahā Śvāsa
c) Tamaka Śvāsa
d) None of the above

9. मत्तर्षभ इवानिशम् is the feature of

a) Chinna Śvāsa
b) Mahā Śvāsa
c) Tamaka Śvāsa
d) None of the above

10. विभ्रान्ताक्ष is the feature of

a) Chinna Śvāsa
b) Mahā Śvāsa
c) Ūrdhva Śvāsa
d) Tamaka Śvāsa

Answer Keys

1	a	2	c	3	c	4	c	5	c
6	b	7	a	8	b	9	b	10	c

Short Answer Questions

1. Describe Tamaka Śvāsa
2. Describe Mahā Śvāsa
3. Describe Chinna Śvāsa
4. Describe Ūrdhva Śvāsa
5. Differentiate the Pañca Śvāsa

Long Answer Questions

1. Explain Śvāsa in detail
2. Explain Nidāna, Samprāpti, Bheda and Lakṣaṇās of Tamaka Śvāsa

◆

TOPIC 14

Kāsa

Introduction

Kāsa is one of the diseases of Prāṇavaha srotas. The disease Kāsa has been named on the basis of its Pratyātma Lakṣaṇa (cardinal symptom). It may occur as a separate disease or as a symptom in other diseases. Ācārya Caraka and Suśruta explained Kāsa after Hikkā and Śvāsa because of their similarity in Nidāna and Cikitsā. But Vāgbhaṭa explained Kāsa along with Raktapitta due to the acuteness of its manifestation and also Raktapitta (haemoptysis) is a complication of an untreated Kāsa.

Nirūkti and Paribhāṣā

- कसृ शब्द कुत्सायाम्॥ (Amarakośa)
- कासः कुशब्दः । (SKD)
- कासः कुत्सित शब्दः॥ (Vācaspatyam)

The term Kāsa is derived from the root 'Kasṛ', meaning "hard," which refers to Kutsāyām (contempt). Hence, Kāsa implies the production of unpleasant or contemptuous sounds.

- कासतेऽनेन कासः॥ (Vācaspatyam)
- कसनात् कासः' इति । (Ca.Ci.18/8)
- कासनं कास इति । (Ma.M.N.11/1-2)
- कसनं कासः । (Āḍ.Śā.P.7/21)

Due to presence of symptom Kāsa (cough), it is known as Kāsa.

- कसति शिरःकण्ठादूर्ध्वं गच्छति वायुरिति कासः, 'कस' गतौ इत्यस्मात्; 'कसनात् कासः । (Ma.M.N.11/1-2)

The word Kāsa is derived from dhātu "Kasa" which refers to move or approach and the word Kasana refers to Kāsa (cough). Pathologically, in the disease Kāsa, the vitiated Vāta forcibly comes out with upward movement through the channels situated in the head and throat by producing a peculiar sound.

Paryāya:

- कासस्तु क्षवथुः॥ (Amara.Manuṣyavarga.3/52)
- कासः क्षवथुरुच्यते॥ (Rā.Ni.Rogādivarga.20/3)

In Amarakośa and Rāja nighaṇṭu, Kṣavathu is considered as the synonym of Kāsa.

Sāmānya Nidāna and Saṃprāpti

- धूमोपघाताद्रजसस्तथैव व्यायामरूक्षान्ननिषेवणाच्च । विमार्गगत्वादपि भोजनस्य वेगावरोधात् क्षवथोस्तथैव ॥ प्राणो ह्युदानानुगतः प्रदुष्टः सम्भिन्नकांस्यस्वनतुल्यघोषः । निरेति वक्त्रात् सहसा सदोषः कासः स विद्वद्भिरुदाहृतस्तु ॥ (Su.Ū.52/4-5)

Dhūmopaghātadrajas (inhalation of smoke, fumes and dust particles), Vyāyāma (physical exercise), Rūkṣānna niṣevaṇā (intake of non-unctuous food), Vimārgagatvādapi bhojana (the food entering into wrong passage or track other than respiratory), Vegāvrodhāt kṣavathostathaiva (suppression of natural urges like sneezing etc.), the Prāṇa Vāyu in association with Udāna Vāyu gets aggravated and a loud noise similar to that of Kāṃsya ghoṣaḥ (broken brass vessel sound) comes out of the mouth suddenly with doṣas. This condition is called Kāsa by the scholars.

- अधःप्रतिहतो वायुरूर्ध्वस्रोतःसमाश्रितः। उदानभावमापन्नः कण्ठे सक्तस्तथोरसि॥ आविश्य शिरसः खानि सर्वाणि प्रतिपूरयन्। आभञ्जन्नाक्षिपन् देहं हनुमन्ये तथाऽक्षिणी॥ नेत्रे पृष्ठमुरःपार्श्वे निर्भुज्य स्तम्भयंस्ततः। शुष्को वा सकफो वाऽपि कसनात्कास उच्यते॥ (Ca.Ci.18/6-8)

Vāta obstructed in the lower parts of the body, moves upwards and gets accumulated in the upper part of the body, taking control over the functions of Udāna Vāyu gets adhered to the region of throat and chest. Then, this Vāta enters in all vacant spaces of the head, fills them and produces Ābhañjana (breaking pain) and Ākṣepaka (convulsive movements) of the Deha (body), Hanu (jaws), Manya (neck) and Akṣi (eyes). Then this obstructed Vāta causes Nirbhujya (bend awry) and Sthambha (stiffness) in the Netra (eyes), Pṛṣṭha (back), Uras (chest) and Pārśva (flanks) and further produces Śuṣkā Kāsa (dry cough) or Sakapha Kāsa (cough with phlegm). Since there is Kasana (cough), it is called Kāsa.

- हेतुभेदात्प्रतीघातभेदो वायोः सरंहसः॥ यद्रुजाशब्दवैषम्यं कासानां जायते ततः। (A.H.Ni.3/21)
- प्रतिघातविशेषेण तस्य वायोः सरंहसः। वेदनाशब्दवैशिष्ट्यं कासानामुपजायते॥ (Ca.Ci.18/9)

The Vāta which is impetuous in nature gets obstructed by specific causes (covering by Kapha etc.) produces specific pain and sound according to the specific location.

Samprāpti Ghaṭaka of Kāsa:

Doṣa	Vāta, Kapha
Dūṣya	Rasa
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa and Prāṇa vaha
Srotoduṣṭi	Saṅga, Vimārga gamana
Sañcāra sthāna	Rasavāhinyaḥ
Udbhava sthāna	Āmāśaya
Vyakta sthāna	Vaktra
Adhiṣṭhāna	Uras
Rogamārga	Abhyantara

Pūrvarūpa of Kāsa

- ⊙ पूर्वरूपं भवेत्तेषां शूकपूर्णगलास्यता। कण्ठे कण्डूश्च भोज्यानामवरोधश्च जायते॥ (Ca.Ci.18/5)

Śūkapūrṇagalasya (feeling as if mouth and throat are filled with thorns), Kaṇṭhe kaṇḍū (itching sensation in the throat) and Bhojyānāmavarodha (obstruction in swallowing the food) are the premonitory symptoms of Kāsa.

- ⊙ भविष्यतस्तस्य तु कण्ठकण्डूर्भोज्योपरोधो गलतालुलेपः । स्वशब्दवैषम्यमरोचकोऽग्निसादश्च लिङ्गानि भवन्त्यमूनि। (Su.U.52/7)

- ⊙ तेषां भविष्यतां रूपं कण्ठे कण्डूररोचकः॥ शूकपूर्णाभकण्ठत्वम्..... ॥ (A.H.Ni.3/18-19)

Ācārya Suśruta added Galatālulepaḥ (feeling as if throat and palate are coated), Svaśabdavaiṣamya (alteration of one's own voice), Arocaka (anorexia) and Agni sāda (diminished digestive power).

Kāsa Bheda

- ⊙ वातादिजास्त्रयो ये च क्षतजः क्षयजस्तथा। पञ्चैते स्युर्नृणां कासा वर्धमानाः क्षयप्रदाः॥ (Ca.Ci.18/4)

Kāsa is classified into five types namely, Vātaja, Pittaja, Kaphaja, Kṣataja and Kṣayaja. All these types when increased become fatal.

- ⊙ पञ्च कासाः स्मृता वातपित्तश्लेष्मक्षतक्षयैः॥ क्षयायोपेक्षिताः सर्वे बलिनश्चोत्तरोत्तरम्। (A.H.Ni.3/17-18)
- ⊙ स वातपित्तप्रभवः कफाच्च क्षतात्तथाऽन्यः क्षयजोऽपरश्च ।
पञ्चप्रकारः कथितो भिषग्भिर्विवर्धितो यक्ष्मविकारकृत् स्यात् ॥ (Su.U.52/6)

Kāsa is classified into five types namely, Vātaja, Pittaja, Kaphaja, Kṣataja and Kṣayaja. All these types of Kāsa lead to Kṣaya or Yakṣma if they become chronic or neglected and are strong in the succeeding order

Doṣaja Kāsa Lakṣaṇās

Vātaja Kāsa

- ⊙ रूक्षशीतकषायाल्पप्रमितानशनं स्त्रियः। वेगधारणमायासो वातकासप्रवर्तकाः॥
हृत्पार्श्वोरःशिरःशूलस्वरभेदकरो भृशम्। शुष्कोरःकण्ठवक्रस्य हृष्टलोम्नः प्रताम्यतः॥
निर्घोषदैन्यस्तननदौर्बल्यक्षोभमोहकृत्। शुष्ककासः कफं शुष्कं कृच्छ्रान्मुक्त्वाऽल्पतां व्रजेत्॥
स्निग्धाम्ललवणोष्णैश्च भुक्तपीतैः प्रशाम्यति। ऊर्ध्ववातस्य जीर्णेऽन्ने वेगवान्मारुतो भवेत्॥ (Ca.Ci.18/10-13)

Indulging in Rukṣa (non-unctuous), Śīta (cold) and Kaṣāya (astringent) food substances, Alpapramitānaśanaṃ (taking food in less quantity or limited quantity or not taking food), Striyaḥ (excessive indulgence with women), Vegadhāraṇa (suppressing natural urges) and Āyāsa (excessive exertion) produces Vātaja Kāsa.

Hṛtpārśvoraḥśiraḥ śūla (excruciating pain in the cardiac region, flanks, chest and head), Svarabhedakara bhṛśam (severe hoarseness of voice), Śuṣkoraḥkaṇṭhavaktra (dryness of chest, throat and mouth), Hṛṣṭalomnaḥ (horripilation), Pratāmyataḥ (feeling as if entering in darkness or blackout), Nirghoṣa (soundless or feeble sound), Dainya (distress), Daurbalya (weakness), Kṣobha (irritation), Mohakṛt (delusion), Śuṣkakāsaḥ (dry cough), Kaphaṃ śuṣkaṃ kṛcchrānmuktvāalpatāṃ vrajet (the paroxysm of cough diminishes after expectoration of dry phlegm with difficulty) are the signs and symptoms of Vātaja Kāsa. And it gets pacified with Snigdhāmlalavaṇoṣṇaiśca bhuktapītaiḥ praśāmyati (diet and drinks which are unctuous, sour, salted and hot) and Ūrdhvavātasya jīrṇeanne vegavānmāruto bhavet (the upward movement of Vāta becomes flares up after the digestion of food).

- ⊙ हृच्छङ्खमूर्धोदरपार्श्वशूली क्षामाननः क्षीणबलस्वरौजाः । प्रसक्तमन्तःकफमीरणेन कासेत्तु शुष्कं स्वरभेदयुक्तः ॥ (Su.U.52/8)

The clinical features of Vātaja Kāsa are Hṛcchaṅkhamūrdhodarapārśva śūla (pain in the cardiac region, temporal region, head, abdomen and flanks), Kṣāmānanaḥ (anxious face), Kṣīṇabalasvaraujāḥ (decreased strength, voice and Ojas) and Prasaktamantaḥkaphamīraṇena kāsettu śuṣkaṃ (even though the chest is full of Kapha, the Vātaja kāsa is dry) and it is associated with Svarabheda (hoarseness of voice).

Pittaja Kāsa

⦿ कटुकोष्णविदाह्यम्लक्षाराणामतिसेवनम्। पित्तकासकरं क्रोधः सन्तापश्चाग्निसूर्यजः॥
पीतनिष्ठीवनाक्षित्वं तिक्तास्यत्वं स्वरामयः। उरोधूमायनं तृष्णा दाहो मोहोऽरुचिर्भ्रमः॥
प्रततं कासमानश्च ज्योतींषीव च पश्यति। श्लेष्माणं पित्तसंसृष्टं निष्ठीवति च पैत्तिके॥ (Ca.Ci.18/14-16)

Excessive indulgence in Kaṭu (non-unctuous), Uṣṇa (cold), Vidāhī (produces burning sensation with indigestion), Amla (sour) and Kṣārā (alkaline) food substances, Krodhaḥ (anger), and Santāpaścāgnisūryajaḥ (heat of fire and sun) are the causes of Pittaja Kāsa.

Pītaniṣṭhīvanākṣitvaṃ (yellowish sputum and eyes), Tiktāsyatvaṃ (bitter taste in the mouth), Svarāmayaḥ (disorders of voice), Urodhūmāyanaṃ (feeling as if smoke is coming out from chest), Tṛṣṇā (thirst), Dāha (burning sensation), Moha (delusion), Aruci (anorexia), Bhrama (giddiness), Pratataṃ kāsamānaśca jyotīṃṣīva ca paśyati (while coughing constantly visualizes shining stars) and Śleṣmāṇāṃ pittasaṃsṛṣṭaṃ niṣṭhīvati (expectorates phlegm mixed with pitta) are the signs and symptoms of Pittaja Kāsa.

⦿ उरोविदाहज्वरवक्त्रशोषैरभ्यर्दितस्तिक्तमुखस्तृषार्तः । पित्तेन पीतानि वमेत् कटूनि कासेत् स पाण्डुः परिदह्यमानः ॥
(Su.U.52/9)

The clinical features of Pittaja Kāsa are Urovidāha (burning sensation in the chest), Jvara (fever), Vaktraśoṣa (dryness of the mouth), Tiktamukha (bitter taste in the mouth), Tṛṣārtaḥ (feels thirsty), Pītāni vamet kaṭūni kāset (yellowish and acrid vomitus), Sapāṇḍuḥ (pallor) and Paridahyamānaḥ (burning sensation all over the body).

Kaphaja Kāsa

⦿ गुर्वभिष्यन्दिमधुरस्निग्धस्वप्नाविचेष्टनैः। वृद्धः श्लेष्माऽनिलं रुद्ध्वा कफकासं करोति हि॥
मन्दाग्नित्वारुचिच्छर्दिपीनसोत्क्लेशगौरवैः। लोमहर्षास्यमाधुर्यक्लेदसंसदनैर्युतम्॥
बहुलं मधुरं स्निग्धं निष्ठीवति घनं कफम्। कासमानो ह्यरुग् वक्षः सम्पूर्णमिव मन्यते॥ (Ca.Ci.18/17-19)

Excessive indulgence in Guru (heavy), Abhiṣyandi (slimy), Madhura (sweet) and Snigdha (unctuous) food substances, Divasvapna (day sleep), Viceṣṭanai (absence of activities and Vṛddhaḥ śleṣmāanilaṃ ruddhavā (aggravated Kapha obstructing the Vāta) produces Kaphaja Kāsa.

Mandāgnitva (decreased digestive power), Aruci (anorexia), Chardi (vomiting), Pīnasa (coryza), Utkleśa (nausea), Gauravaiḥ (heaviness), Lomaharṣa (horripilation), Āsyamādhurya (sweet taste in the mouth), Kleda (moistening of body parts), Saṃsadana (weakness), Bahulaṃ madhuraṃ snigdhaṃ niṣṭhīvati ghanaṃ kapham (expectorates copious, thick, sweet, unctuous and thick phlegm) and Kāsamāno hi aruk vakṣaḥ saṃpūrṇamiva manyate (while coughing feels that his entire chest is filled with Kapha and has no pain) are the signs and symptoms of Kaphaja Kāsa.

⦿ प्र(वि)लिप्यमानेन मुखेन सीदन् शिरोरुजार्तः कफपूर्णदेहः । अभक्तरुग्गौरवसादयुक्तः कासेत ना सान्द्रकफं कफेन ॥
(Su.U.52/10)

The clinical features of Kaphaja Kāsa are Pralipyamānena mukhena sīdan (coating of the mouth), Śirorujārtaḥ (head ache), Kaphapūrna dehaḥ (excess of Kapha in the body), Abhakta (loss of appetite), Ruk (pain), Gaurava (heaviness), Sāda (malaise) and Kāset sāndrakaphaṃ (expectorates thick sputum).

Differentiate Doṣaja Kāsa

Lakṣaṇa	Vātaja Kāsa	Pittaja Kāsa	Kaphaja Kāsa
Śūla	Hṛtpārśvoraḥśiraḥ śūla	Urovidāha	Śirorujārtaḥ
Svara	Svarabhedakara bhṛśam	Svarāmayaḥ	-
Cetas	Kṣobha, Moha	Moha	-
Āsya	Śuṣkoraḥkaṇṭhavaktra	Tiktāsyatvaṃ, Vaktraśoṣa	Āsyamādhurya
Kapha Niṣṭhivana	Kaphaṃ śuṣkaṃ krcchrānmuktvāalpatāṃ vrajet	Pītaniṣṭhīvana	Bahulaṃ madhuraṃ snigdhaṃ niṣṭhīvati ghanaṃ kapham
Anubandha Lakṣaṇa	Hṛṣṭalomnaḥ, Pratāmyataḥ , Nirghoṣa, Dainya, Daurbalya, Kṣāmānanaḥ, Kṣīṇabalasvaraujāḥ	Jvara, Urodhūmāyanaṃ, Tṛṣṇā, Dāha, Aruci, Bhrama, Sapāṇḍuḥ	Mandāgnitva, Aruci, Chardi, Pīnasa, Utkleśa, Gauravaiḥ, Lomaharṣa, Kleda, Saṃsadana

Kṣataja Kāsa

⦿ वक्षोऽतिमात्रं विहतं तु यस्य व्यायामभाराध्ययनाभिघातैः । विश्लिष्टवक्षाः स नरः सरक्तं ष्ठीवत्यभीक्ष्णं क्षतजं तमाहुः ॥
(Su.U.52/11)

Vihata Vakṣa (ulceration in the chest) caused by excessive indulgence in Vyāyāma (exercise), Bhāra (carries heavy weight), Adhyayana (loud reading) and Abhighāta (injuries) thereon affects the chest and gives rise to Saraktaṃ ṣṭhivana (expectoration of blood during cough - haemoptysis). This condition is known as Kṣataja Kāsa.

⦿ अतिव्यवायभाराध्वयुद्धाश्वगजविग्रहैः। रूक्षस्योरः क्षतं वायुर्गृहीत्वा कासमावहेत्॥
स पूर्वं कासते शुष्कं ततः ष्ठीवेत् सशोणितम्। कण्ठेन रुजताऽत्यर्थं विरुग्णेनेव चोरसा॥
सूचीभिरिव तीक्ष्णाभिस्तुद्यमानेन शूलिना। दुःखस्पर्शेन शूलेन भेदपीडाभितापिना॥
पर्वभेदज्वरश्वासतृष्णावैस्वर्यपीडितः। पारावत इवाकूजन् कासवेगात्क्षतोद्भवात्॥ (Ca.Ci.18/20-23)

Excessive indulgence in Vyavāya (sexual intercourse), Bhāra (carries heavy weight), Adhva (walking long distance) Yuddhā (fighting) and Aśvagajavigraiḥ (struggles with horse and elephant) etc., factors produces Rūkṣata (dryness of the body) and Uraḥkṣata (injury to his chest); this leads to aggravation of Vāta there by resulting in manifestation of the disease Kṣataja Kāsa.

Sa pūrvaṃ kāsate śuṣkaṃ tataḥṣṭhīvet saśoṇitam (initially there will be dry cough, but later it starts expectorating blood tinged phlegm), Kaṇṭhena rujatāatyarthaṃ (severe throat pain), Virugṇeneva ca urasā (feels as if the chest is cracked), Sūcībhiriva tīkṣṇābhistudyamānena śūlinā (pricking type of pain as if pricked by sharp needles), Duḥkhasparśena śūlena bhedapīḍābhitāpinā (miserably seized with tenderness, excruciating and piercing pain), Parvabheda (joint pains), Jvara (fever), Śvāsa (dyspnoea), Tṛṣṇā (thirst), Vaisvarya pīḍitaḥ (altered voice) and Pārāvata ivākūjan kāsavegāt (cooing sound like pigeon while coughing) are the signs and symptoms of Kṣataja Kāsa.

Differential diagnosis of Kṣataja Kāsa and Kṣata Kṣīṇa

Lakṣaṇa	Kṣataja Kāsa	Kṣata Kṣīṇa
Śūla	Kaṇṭhena rujatāatyarthaṃ, Virugṇeneva ca urasā	Urovirujyate tasya bhidyateatha vibhajyate, Pārśve prapīḍyate
Svara	Vaisvarya pīḍitaḥ	Svarāmayaḥ
Śabda	Pārāvata ivākūjan kāsavegāt	Kāsa
Kapha Niṣṭhivana	Sa pūrvaṃ kāsate śuṣkaṃ tataḥṣṭhīvet saśoṇitam	Duṣṭaḥ śyāva sudurgandhaḥ pīto vigrathito bahuḥ kāsamānasya ca śleṣmā saraktaḥ saṃpravartate
Anubandha Lakṣaṇa	Parvabheda, Jvara, Śvāsa , Tṛṣṇā, Vaisvarya pīḍitaḥ and Pārāvata ivākūjan kāsavegāt	Śuṣyatyaṅga, Pravepate, Kramādvīryaṃ balaṃ varṅo ruciragniśca hīyate, Jvara, Vyathā, Manodainya, Viḍbheda, Agnivadhādapi
Upadrava	Rājayakṣmā	Atyartha Kṣīṇa, Rājayakṣmā

Kṣayaja Kāsa

- ⊙ विषमासात्म्यभोज्यातिव्यवायाद्वेगनिग्रहात्। घृणिनां शोचतां नृणां व्यापन्नेऽग्नौ त्रयो मलाः॥
कुपिताः क्षयजं कासं कुर्युर्देहक्षयप्रदम्। दुर्गन्धं हरितं रक्तं ष्ठीवेत् पूयोपमं कफम्॥
स्थानादुत्कासमानश्च हृदयं मन्यते च्युतम् । अकस्मादुष्णशीतार्तो बह्वाशी दुर्बलः कृशः॥
स्निग्धाच्छमुखवर्णत्वक् श्रीमद्दर्शनलोचनः । पाणिपादतलैः श्लक्ष्णैः सततासूयको घृणी॥
ज्वरो मिश्राकृतिस्तस्य पार्श्वरुक् पीनसोऽरुचिः। भिन्नसंहतवर्चस्त्वं स्वरभेदोऽनिमित्ततः॥
इत्येष क्षयजः कासः ... ॥ (Ca.Ci.18/24-29)

Indulging in Viṣamāsātmyabhojana (irregular dietary habits and unwholesome foods), Ativyavāya (excessive sexual intercourse), Vegānigrahāt (suppression of natural urges), Ghṛṇināṃ (violent) and Śocatāṃ (grief) factors affects the Agni resulting in aggravation of three doṣas and produces the disease Kṣayaja Kāsa.

Durgandhaṃ haritaṃ raktaṃ ṣṭhīvet pūyopamaṃ kaphaṃ (expectoration of phlegm are putrid, greenish and reddish in colour associated with pus), Sthānādutkāsamānaśca hṛdayaṃ manyate cyutam (feels as if the heart is displaced while coughing), Akasmāduṣṇaśītārtā (suddenly feels heat and cold sensations), Bahvāśī (becomes excessive eater), Durbalaḥ kṛśaḥ (becomes weak and emaciated), Snigdhācchamukhavarṇatvak (face, complexion and skin becomes clean and unctuous), Śrīmaddarśanalocanaḥ (glowing eyes), Pāṇi pāda talaiḥ ślakṣṇaiḥ (palm and sole become smooth), Satatāsūyako ghṛṇī (always envious), Jvara miśrākṛtita (mixed characters of fever), Pārśvaruk (pain in the flanks), Pīnasa (coryza), Aruci (anorexia), Bhinnasaṃhatavarcastvaṃ (passes liquid or solid faeces) and Svarabhedoanimittataḥ (hoarseness of voice without any cause) are the signs and symptoms of Kṣayaja Kāsa.

- ⊙ स गात्रशूलज्वरदाहमोहान् प्राणक्षयं चोपलभेत कासी । शुष्यन् विनिष्ठीवति दुर्बलस्तु प्रक्षीणमांसो रुधिरं सपूयम् ।
ससर्वलिङ्गं भृशदुश्चिकित्स्यं चिकित्सितज्ञाः क्षयजं वदन्ति ॥ (Su.U.52/12-13)

Gātraśūla (body ache), Jvara (fever), Dāha (burning sensation), Moha (delusion) and Prāṇakṣayaṃ (decrease of vitality), Śuṣyan (emaciation of body), Durbalastu (weakness), Prakṣīṇamāṃso (loss of muscles) and Viniṣṭhivati rudhiraṃ sapūyam (spitting blood streaked with pus) are the symptoms of Kṣayaja Kāsa. Due to intensive action of all the three doṣas, this type of Kāsa is Duścikitsyaṃ (difficult to be cured).

Differential diagnosis of Kāsa based on Kapha Lakṣaṇa

Kāsa	Kapha Lakṣaṇa
Vātaja	Kaphaṃ śuṣkaṃ kṛcchrānmuktvāalpatāṃ vrajet (the paroxysm of cough diminishes after expectoration of dry phlegm with difficulty)
Pittaja	Pītaniṣṭhīvana (yellowish sputum)
Kaphaja	Bahulaṃ madhuraṃ snigdhaṃ niṣṭhīvati ghanaṃ kapham (expectorates copious, thick, sweet, unctuous and thick phlegm)
Kṣataja	Sa pūrvaṃ kāsate śuṣkaṃ tataḥṣṭhīvet saśoṇitam (initially there will be dry cough, but later it starts expectorating blood tinged phlegm)
Kṣayaja	Durgandhaṃ haritaṃ raktaṃ ṣṭhīvet pūyopamaṃ kaphaṃ (expectoration of phlegm are putrid, greenish and reddish in colour associated with pus)

Differential diagnosis of Kṣayaja Kāsa and Rājayakṣmā

Lakṣaṇa	Kṣayaja Kāsa	Rājayakṣmā
Nidāna	Bahukāraṇa such as Viṣamāsātmyabhojana, Ativyavāya, Vegānigrahāt, Ghṛṇināṃ and Śocatāṃ	Catur Nidāna i.e. Sāhasa, Vegarodhāt, Kṣaya and Viṣamāśanāt
Doṣa	Vāta Kapha	Kapha pradhāna Tridoṣa
Dūṣyā	Rasa and Rakta dhātu	Saptadhātu and Mala
Srotas	Anna and Prāṇavaha Srotas	Rasa, Anna, Śukravaha and later all the Srotas

Lakṣaṇa	Kṣayaja Kāsa	Rājayakṣmā
Lakṣaṇa	Durgandhaṃ haritaṃ raktaṃ ṣṭhīvet pūyopamaṃ kaphaṃ, Sthānādutkāsamānaśca hṛdayaṃ manyate cyutam, Akasmāduṣṇaśītārtā, Bahvāśī, Durbalaḥ kṛśaḥ, Pīnasa, Aruci, Snigdhācchamukhavarṇatvak, Svarabhedoanimittataḥ, Satatāsūyako ghṛṇī, Śrīmaddarśanalocanaḥ, Bhinnasaṃhatavarcastvaṃ	Trirūpa Ṣaḍrūpa Ekadaśarūpa
Upadravā	Rājayakṣmā	Kaṇṭhoddhvaṃsa, Uroruja, Jṛmbha, Aṅgamarda, Niṣṭhīva, Agnisāda and Āsyapūtitāḥ
Sādhyāsādhyata	Duścikitsyaṃ	Cikitsāvarjya

Sādhyāsādhyata of Kāsa

- ◉ त्रीन्साध्यान्साधयेत्पूर्वान् पथ्यैर्याप्यांश्च यापयेत्। (Ca.Ci.18/31)

The first three types viz. Vātaja, Pittaja and Kaphaja Kāsa are Sādhya (curable); Kṣayaja and Kṣataja Kāsa are Yāpya (manageble with specific medications and diet protocol).

- ◉ क्षयजः कासः क्षीणानां देहनाशनः। साध्यो बलवतां वा स्याद्याप्यस्त्वेवं क्षतोत्थितः॥
  नवौ कदाचित् सिध्येतामेतौ पादगुणान्वितौ। स्थविराणां जराकासः सर्वो याप्यः प्रकीर्तितः॥ (Ca.Ci.18/29-30)

Kṣayaja Kāsa is Dehanāśanaḥ (fatal) in cases of Kṣīṇā (emaciated) and it is Sādhya (curable) in Balavatāṃ (strong person). Kṣataja Kāsa is Yāpya (palliable) in Balavatāṃ (strong persons). Both Kṣayaja and Kṣataja Kāsa are Sādhya (curable) if it is Nava (recent origin), Sidhyetāmetau Pādaguṇānvitau (endowed with perfect four limbs of treatment). However, all types of Kāsa are Yāpya (palliable) in Jarā (aged persons).

- ◉ वृद्धत्वमासाद्य भवेत्तु यो वै याप्यं तमाहुर्भिषजस्तु कासम् ॥ (Su.Ū.52/13)

Scholars described that all types of Kāsa in an old man has been considered as Yāpya (palliable) due to degeneration of tissues in aging.

- ◉ कासाच्छ्वासक्षयच्छर्दिस्वरसादादयो गदाः। भवन्त्युपेक्षया यस्मात्तस्मात्तं त्वरया जयेत्। (A.H.Ni.3/38)

If Kāsa is not treated properly, it give rises to the diseases like Śvāsa (dyspnoea), Kṣaya (consumption), Chardi (vomiting) and Svarasaḥda (loss of voice) etc., hence it should be treated promptly.

In Contemporary science,

Kāsa cannot be correlated with a single disease in contemporary science as it is rather seen as a symptom in many diseases than a disease itself. Cough is commonly seen as a symptom in a diseases caused by various kind of infections and inflammations of respiratory tracts and sinus, obstructive pathology of lungs, seasonal variation, smoking, acid peptic disorders, chest injury and even in carcinoma.

MCQ's

1. Doṣa Predominance in Kāsa
   a) Vātakapha b) Vātapitta
   c) Kaphapitta d) Sannipātaja
2. Kāsa is of .... types
   a) 2 b) 4
   c) 5 d) 6
3. Pūrvarūpa of Kāsa is
   a) Galatālulepaḥ b) Kaṇṭhe kaṇḍū
   c) Svaśabdavaiṣamya d) All of the above
4. शूकपूर्णगलास्यता is the Pūrvarūpa of
   a) Hikkā b) Śvāsa
   c) Kāsa d) Galagaṇḍa
5. शुष्कोरःकण्ठवक्त्र is the feature of
   a) Vātaja Kāsa b) Pittaja Kāsa
   c) Kaphaja Kāsa d) Kṣataja Kāsa
6. Pārāvata ivākūjan is the feature of
   a) Vātaja Kāsa b) Pittaja Kāsa
   c) Kaphaja Kāsa d) Kṣataja Kāsa
7. स पूर्वं कासते शुष्कं ततः ष्ठीवेत् सशोणितम् is the feature of
   a) Kṣayaja Kāsa b) Kṣataja Kāsa
   c) Kaphaja Kāsa d) Rājayakṣmā
8. श्रीमद्दर्शनलोचनः is the feature of
   a) Kṣayaja Kāsa b) Kṣataja Kāsa
   c) Kaphaja Kāsa d) Rājayakṣmā

9. दुर्गन्धं हरितं रक्तं ष्ठीवेत् पूयोपमं कफम् is the feature of
   - a) Vātaja Kāsa
   - b) Pittaja Kāsa
   - c) Kṣataja Kāsa
   - d) Kṣayaja Kāsa

10. Kṣataja Kāsa is
   - a) Sādhya
   - b) Kṛcchrā Sādhya
   - c) Yāpya
   - d) Asādhya

Answer Keys

1	a	2	c	3	d	4	c	5	a	6	d	7	b	8	a	9	d	10	c

Short Answer Questions

1. Explain the Nidāna and Saṃprāpti of Kāsa
2. Explain the types of Kāsa
3. Explain Kṣataja Śvāsa
4. Explain Kṣayaja Śvāsa
5. Write the difference between Kṣayaja Kāsa and Rājayakṣma
6. Write the difference between Kṣayaja Kāsa and Kṣata Kṣīṇa

Long Answer Questions

1. Define Kāsa and explain Nidāna, Saṃprāpti, Bheda and Lakṣaṇās of Kāsa

◆

TOPIC 15

Rājayakṣmā and Śoṣa

Introduction

Rājayakṣmā is a Tridoṣaja Vyādhi affecting the Madhyama rogamārga and it appears to be depleting the Saptadhātus and is considered as Mahāroga. Ācārya Caraka described Rājayakṣmā after Kuṣṭha cikitsā because of the involvement of Tridoṣas in both the diseases and Ācārya Cakrapāṇi considered it as Mahāroga. The untreated Kāsa produces Śvāsa and Kṣaya; hence Ācārya Vāgbhaṭa described Rājayakṣmā after Hikkā and Śvāsa. Ācārya Suśruta described it after Atisāra because Rājayakṣmā manifest as a consequence of Atisāra or Jvarātisāra and he considered it as Aupasargika roga and Ācārya Ḍalhaṇa included Rājayakṣmā under Ādibala Pravṛtta roga. This disease first afflicted the Candrā (Moon - the King of Nakṣatras and Dvijā), hence it is named as Rājayakṣmā.

Nirūkti and Paribhāṣā of Rājayakṣmā

- ◉ यदा कष्टतमत्वात्तदा 'राजेव यक्ष्मा राजयक्ष्मेति' ॥ (Ca.Ca.Ni.6/12)
- ◉ राजा यक्ष्मा राजयक्ष्मा इति॥ (SKD)
- ◉ यक्ष्मणां राजा राजयक्ष्मा" इत्युक्तम् ॥ (Ma.M.N.10/1)
- ◉ यक्ष्मा रोगो रोगाणां राजा अतो राजयक्ष्मेति ॥ (Ḍa.Su.U.41/4-5)

The term Rājayakṣmā is composed of two words Rāja and Yakṣmā. The word 'Rāja' means 'Dīptau' which means the person who rules and the word 'Yakṣmā' means 'Roga' which means disease. Hence Rājayakṣmā means King among diseases (Rājeva yakṣmā) or King's (Moon - the King of Nakṣatras and Dvijā) disease which is most troublesome.

- ◉ राज्ञश्चन्द्रस्य क्षयकारका यक्ष्मा राजा चासौ यक्ष्मा चेति वा । (SKD)
- ◉ यस्मात् स राज्ञः प्रागासीद्राजयक्ष्मा ततो मतः ॥ (Ca.Ci.8/11)

- ⊙ राज्ञश्चन्द्रमसो यस्मादभूदेष किलामयः । तस्मात्तं राजयक्ष्मेति केचिदाहुः पुनर्जनाः ॥ (Su.U.41/5)
- ⊙ यक्ष्माणां राजा राजयक्ष्मा यद्वा चन्द्रमासो जातत्वात् राजयक्ष्मेति॥ (Kā.Śā.P.7/22-23)

Since the disease Yakṣmā first afflicted the King Candra (Moon- King of planets), the learned scholars call it Rājayakṣmā.

- ⊙ यक्ष्मशब्देन च राजयक्ष्मवदनेकरोगयुक्तत्वं विकाराणां दर्शयति; ॥ (Ca.Ca.Ni.1/5)

The word Yakṣmā indicates Anekarogayuktatvaṃ vikārāṇāṃ (the disease associated with several disorders), like Rājayakṣmā.

- ⊙ राजयक्ष्मा रोगसमूहानां ॥ (Ca.Sū.25/40)

  Rājayakṣmā is an aggregates or collection or group of diseases

- ⊙ अनेकरोगानुगतो बहुरोगपुरोगमः। राजयक्ष्मा क्षयः शोषो रोगराडिति च स्मृतः ॥ (A.H.Ni.5/1)
- ⊙ राजयक्ष्मा क्षयः शोषो रोगराटिति च स्मृतः॥ (SKD)

Rājayakṣmā follows many diseases and many diseases arise from it. Kṣaya, Śoṣa and Rogarāṭ are the synonyms of the disease Rājayakṣmā.

- ⊙ रसः स्रोतःसु रुद्धेषु स्वस्थानस्थो विदह्यते । स ऊर्ध्वं कासवेगेन बहुरूपः प्रवर्तते॥
  जायन्ते व्याधयश्चातः षडेकादश वा पुनः। येषां सङ्घातयोगेन राजयक्ष्मेति कथ्यते ॥ (Ca.Ci.8/43-44)

Due to obstruction in the Rasavāhi srotas, the Rasa dhātu gets accumulated in its own place increases and then comes out with force of coughing from upper orifices in various forms. Consequently, Ṣaḍrūpa or Ekādaśarūpa are manifested, collection of which is called Rājayakṣmā.

Nirūkti and Paribhāṣā of Śoṣa

- ⊙ रसादिशोषणात् शोषः॥ (Kā.Śā.P.7/22-23)
- ⊙ संशोषणाद्रसादीनां शोष इत्यभिधीयते ॥ (Ma.M.N.10/1)
- ⊙ रसादिशोषणाच्छोषो ॥ (A.H.Ni.5/3)
- ⊙ संशोषणाद्रसादीनां शोष इत्यभिधीयते ॥ (Su.U.41/5)

That which causes desiccation of Rasādi dhātus is known as Śoṣa.

- ⊙ शोषेणेति राजयक्ष्मणा। (Ca.Ca.Ci.4/62-72)

  Śoṣa is also known as Rājayakṣmā.

- ⊙ अनेकरोगानुगतो बहुरोगपुरोगमः । दुर्विज्ञेयो दुर्निवारः शोषो व्याधिर्महाबलः ॥ (Su.U.41/3)

Śoṣa follows many diseases and many diseases arise from it. It is Durvijñeya (hardly conceivable i.e. difficult to be diagnosed) and Durnivāraḥ (invincible i.e. difficult to be cured). *Hence, it is considered as* Mahābalaḥ vyādhi (very powerful or strong disease).

Nirūkti and Paribhāṣā of Kṣaya

- ⊙ क्रियाक्षयकरत्वाच्च क्षय इत्युच्यते पुनः ॥ (Su.U.41/4)

That which produces a loss of functional capacity is called Kṣaya

- ⊙ क्षयात् रसादिशुक्रान्तानां धातूनां क्षयात्। (Ḍa.Su.U.41/8-9)

That which causes depletion of Rasādi dhātus is known as Kṣaya.

- ⊙ क्षयोऽपि राजयक्ष्मसंज्ञः कथ्यते॥ (Āḍ.Śā.P.7/22-23)

Kṣaya is also known as Rājayakṣmā.

- ⊙ क्षीयते अनेनेति क्षय इति। (Kā.Śā.P.7/22-23)
- ⊙ क्षथात् क्षीयतेऽनेनेति क्षयः (SKD)

That which produces depletion is called Kṣaya.

- ⊙ देहौषधक्षयकृतेः क्षयस्तत्सम्भवाच्च सः। (A.H.Ni.5/3)

That which leads to depletion of the strength of body and medicines, is called as Kṣaya.

Nirūkti and Paribhāṣā of Rogarāṭ

- ⊙ रोगराट् तेषु राजनात्। (A.H.Ni.5/3)
- ⊙ बहुषु रोगेषु मध्ये रोगत्वेनैष एव भ्राजते, तस्माद्रोगराडित्युच्यते। (Aru.A.H.Ni.5/3)
- ⊙ [राज]वद्रोगेषु राजत इति रोगराट् । (He.A.H.Ni.5/3)

Among all the diseases, the one which shines or reigns over them is called Rogarāṭ or in other words, prime most disease or king of diseases.

Synonyms of Rājayakṣmā

- ⊙ क्रोधो यक्ष्मा ज्वरो रोग एकार्थो दुःखसञ्ज्ञकः । (Ca.Ci.8/11)
- ⊙ राजयक्ष्मा क्षयः शोषो रोगराडिति च स्मृतः । (A.H.Ni.5/1)

Krodha, Yakṣmā, Jvara, Śoṣa, Kṣaya and Rogarāṭ are the synonyms of Rājayakṣmā.

Bheda of Rājayakṣmā

- ⊙ **Based on the predominance of doṣas:** Rājayakṣmā is classified into four types based on the predominance of doṣas namely Vātaja, Pittaja, Kaphaja and Sannipātaja Rājayakṣmā
- ⊙ **Based on the causative factors:** Rājayakṣmā is classified into four types based on the causative factors namely Sāhasaja, Vega sandhāraṇaja, Kṣayaja and Viṣamāśanaja Rājayakṣmā

- ⊙ **Based on the Dhātu Kṣaya:** Śoṣa is classified into seven types based on the Dhātu Kṣaya namely Vyavāya, Śoka, Vārdhakya, Vyāyāma, Adhva, Upavāsa, Vraṇa and Uraḥkṣata.
- ⊙ **Based on the clinical presentations:** Rājayakṣmā is classified into three types based on the clinical presentations namely Trirūpa, Ṣaḍrūpa and Ekādaśarūpa Rājayakṣmā
- ⊙ **Based on the pathogenesis:** Rājayakṣmā is classified into two types based on the pathogenesis namely Anuloma and Pratiloma Kṣaya.

⊙ एकादशानामेकस्मिन् सान्निध्यात्तन्त्रयुक्तितः क्रियाणामविभागेन प्रागेकोत्पादनेन च ।
एक एव मतः शोषः सन्निपातात्मको ह्यतः ॥ उद्रेकात्तत्र लिङ्गानि दोषाणां निपतन्ति हि ॥ (Su.U.41/6-8)

But Ācārya Suśruta opines that Rājayakṣmā as a single entity, because all the eleven symptoms which are manifested simultaneously with the ushering in of the disease; the line of treatment does not vary irrespective of variation of symptoms and the disease originated first as the effect of Dakṣa prajāpati's curse on the moon was only of one kind. Hence the disease Rājayakṣmā should be considered as single entity though produced due to vitiation of all the three doṣas. Thus, the eleven symptoms are produced due to the predominance of these doṣas.

⊙ केषाञ्चिदेवं शोषो हि कारणैर्भेदमागतः ॥ न तत्र दोषलिङ्गानां समस्तानां निपातनम् ।
क्षया एव हि ते ज्ञेयाः प्रत्येकं धातुसञ्ज्ञिताः ॥ (Su.U.41/26-28)

Some scholars opine that Śoṣa is of different kinds based on the causative factors. But, as all the eleven symptoms produced by vitiation of three doṣas are not fully exhibited, they should be considered to be essentially due to Kṣaya of every single Dhātu.

Sāmānya Nidāna and Saṃprāpti of Rājayakṣmā

⊙ अयथाबलमारम्भं वेगसन्धारणं क्षयम्। यक्ष्मणः कारणं विद्याच्चतुर्थं विषमाशनम्॥ (Ca.Ci.8/13)

⊙ साहसं वेगसंरोधः शुक्रौजःस्नेहसङ्क्षयः। अन्नपानविधित्यागश्चत्वारस्तस्य हेतवः॥ (A.H.Ni.5/4)

Ayatābalamarāmbhaṃ or Sāhasa (physical exertion beyond capacity), Vegasandhāraṇaṃ or Vegasaṃrodhaḥ (suppression of natural urges), Kṣaya or Śukraujaḥ sneha saṃkṣaya (wasting of tissues or depletion of semen, Ojas and Sneha in the body), and Viṣamāśana or Annapānavidhityāga (irregular diet or avoiding the proper regimen of food) are the four-fold causative factors of Rājayakṣmā.

⊙ तैरुदीर्णोऽनिलः पित्तं कफं चोदीर्य सर्वतः। शरीरसन्धीनाविश्य तान् सिराश्च प्रपीडयन्॥
मुखानि स्रोतसां रुद्ध्वा तथैवातिविवृत्य वा। सर्पन्नूर्ध्वमधस्तिर्यग्यथास्वं जनयेद्गदान्॥ (A.H.Ni.5/5-6)

By the above said causes, the aggravated Vāta doṣa causes increase of both Pitta and Kapha and spreads to all the joints of the body and affects Sandhi and Sirās leading to Ruddha (obstruction) or Ativivṛtya (dilatation) of the Srotomukha (orifices of Srotas). Thereafter the aggravated doṣas moves upwards, downwards and sideward and produces various symptoms of Rājayakṣmā depending upon their localization.

⊙ क्षयाद्वेगप्रतीघातादाघाताद्विषमाशनात् ॥ जायते कुपितैर्दोषैर्व्याप्तदेहस्य देहिनः । कफप्रधानैर्दोषैर्हि रुद्धेषु रसवर्त्मसु ॥९॥
अतिव्यवायिनो वाऽपि क्षीणे रेतस्यनन्तरम् । क्षीयन्ते धातवः सर्वे ततः शुष्यति मानवः ॥ (Su.U.41/8-10)

Kṣaya (wasting of tissues), Vegapratighāta (suppression of natural urges), Āghāta (physical exertion beyond capacity) and Viṣamāśana (irregular diet) are the causes, by which the doṣas gets aggravated and spreads to all over the body and produces the disease. Among the aggravated doṣas, Kapha doṣa is the predominant factor having obstructing the Rasavaha srotas which leads to dhātukṣaya of further dhātus (Anuloma kṣaya) or in person whose semen have been exhausted by excessive sexual intercourse leads to dhātukṣaya in the reverse order (Pratiloma Kṣaya) and the person subsequently suffer from the Rājayakṣmā.

⊙ यथास्वेनोष्मणा पाकं शारीरा यान्ति धातवः। स्रोतसा च यथास्वेन धातुः पुष्यति धातुतः॥
स्रोतसां सन्निरोधाच्च रक्तादीनां च सङ्क्षयात्। धातूष्मणां चापचयाद्राजयक्ष्मा प्रवर्तते॥
तस्मिन् काले पचत्यग्निर्यदन्नं कोष्ठसंश्रितम्। मलीभवति तत् प्रायः कल्पते किञ्चिदोजसे॥
तस्मात् पुरीषं संरक्ष्यं विशेषाद्राजयक्ष्मिणः। सर्वधातुक्षयार्तस्य बलं तस्य हि विड्बलम्॥
रसः स्रोतःसु रुद्धेषु स्वस्थानस्थो विदह्यते । स ऊर्ध्वं कासवेगेन बहुरूपः प्रवर्तते॥
जायन्ते व्याधयश्चातः षडेकादश वा पुनः। येषां सङ्घातयोगेन राजयक्ष्मेति कथ्यते॥ (Ca.Ci.8/39-44)

Generally, the dhātus get metabolized by their own ūṣma (dhātvāgnis) and nourished through their respective Srotas. Due to obstruction in these srotas, Raktādi Kṣaya (depletion of Raktādi dhātus) and Dhātūṣmaṇāṃ apacaya (decrease of dhātvāgni) Rājayakṣmā manifests. During this period, whatever food in Koṣṭha digested by Jāṭarāgni is mostly converted into Mala (waste product or faeces) and very little of it is utilized for formation of Ojas. In a state of marked depletion of all the dhātus, the faeces of the patient of Rājayakṣmā should be protected because the strength of the body depends on faeces (Viḍbalaṃ).

Due to obstruction of Rasavaha srotas, the Rasa dhātu gets accumulated in its own place due to Vidagdha (improperly metabolized) and comes out with force coughing through upper orifices in different forms. Subsequently, Ṣaḍrūpa and Ekādaśarūpa are manifested, collection of which is called Rājayakṣmā.

Saṃprāpti Ghaṭaka of Rājayakṣmā

Doṣa	Kapha Pradhāna Tridoṣa
Dūṣya	Sapta dhātu and Mala
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Agni māndhya janya Āma
Srotas	Rasa, Anna, Śukra vaha and later all the Srotas
Srotoduṣṭi	Saṅga, Vimārga gamana

Udbhava sthāna	Āmapakvāśaya
Sañcāra sthāna	Rasavāhini
Vyakta sthāna	Sarva Śārīra
Adhiṣṭhāna	Sarva Śārīra
Rogamārga	Madhyama

Sāhasa Yakṣmā Nidāna, Saṃprāpti and Lakṣaṇās

- युद्धाध्ययनभाराध्वलङ्घनप्लवनादिभिः। पतनैरभिघातैर्वा साहसैर्वा तथाऽपरैः॥
अयथाबलमारम्भैर्जन्तोरुरसि विक्षते। वायुः प्रकुपितो दोषावुदीर्योभौ प्रधावति॥
स शिरःस्थः शिरःशूलं करोति गलमाश्रितः। कण्ठोद्ध्वंसं च कासं च स्वरभेदमरोचकम्॥
पार्श्वशूलं च पार्श्वस्थो वर्चोभेदं गुदे स्थितः। जृम्भां ज्वरं च सन्धिस्थ उरःस्थश्चोरसो रुजम्॥
क्षणनादुरसः कासात् कफं ष्ठीवेत् सशोणितम्। जर्जरेणोरसा कृच्छ्रमुरःशूलातिपीडितः॥
इति साहसिको यक्ष्मा रूपैरेतैः प्रपद्यते। एकादशभिरात्मज्ञो भजेत्तस्मान्न साहसम्॥ (Ca.Ci.8/14-19)

Indulging in Yuddha (battle), Adhyayana (reading with loud voice), Bhāra (weight-lifting), Adhva (walking long distance), Laṅghana (fasting), Plavanādi (swimming etc.), Patana (falling down from height), and Abhighāta (trauma) exerting beyond his capacity or exerts himself in any other similar actions leads to Uraḥkṣata (chest injury) and Vāta gets vitiated, and afflicts the Pitta and Kapha doṣas spreads all over the body and produces the symptoms depending on the location of the body i.e. in Śiras (head) produces Śiraśūla (headache), in Gala (throat) produces Kaṇṭhodvaṃsaṃ (irritation of the throat), Kāsa (cough), Svarabheda (hoarseness of voice) and Arocaka (anorexia), in Pārśva (flanks) produces Pārśvaśūla (pain in the flanks), in Guda (rectum) produces Varcobheda (diarrhoea), in Sandhi (joints), produces Jṛmbhā (yawning) and Jvara (fever), in Uras (chest) produces Uraso ruja (pain in the chest). Due to injury in the chest, Kāsāt kaphaṃ ṣṭhīvana saśoṇita (on coughing, expectoration of phlegm mixed with blood with difficulty) associated with Uraḥśūlāipīḍitah (severe chest pain) is produced. These are the Ekādaśarūpa of Rājayakṣmā caused by Sāhasa (over exertion).

Vegasandhāraṇaṃ Yakṣmā Nidāna, Saṃprāpti and Lakṣaṇās

- ह्रीमत्त्वाद्वा घृणित्वाद्वा भयाद्वा वेगमागतम्। वातमूत्रपुरीषाणां निगृह्णाति यदा नरः॥
तदा वेगप्रतीघातात् कफपित्ते समीरयन्। ऊर्ध्वं तिर्यगधश्चैव विकारान् कुरुतेऽनिलः॥
प्रतिश्यायं च कासं च स्वरभेदमरोचकम्। पार्श्वशूलं शिरःशूलं ज्वरमंसावमर्दनम्॥
अङ्गमर्दं मुहुश्छर्दिं वर्चोभेदं त्रिलक्षणम्। रूपाण्येकादशैतानि यक्ष्मा यैरुच्यते महान्॥ (Ca.Ci.8/20-23)

Due to Hrīmat (bashfulness), Ghṛṇitvāt (contempt) or Bhayā (fear), if the person suppresses the urge of Vātamūtrapurīṣa (flatus, urine and faeces), the Vāta aggravated due to suppression of urges, propels Kapha and Pitta upwards, obliquely and downwards, then produces the disorders having the features of all the three doṣas such as Pratiśyāya (coryza), Kāsa (cough), Svarabheda (hoarseness of voice), Arocaka (anorexia), Pārśvaśūla (pain in the flanks), Śiraśūla (headache), Jvara (fever), Aṃsāvamardanam (pain in the shoulders), Aṅgamarda (bodyache), Muhuśchardi (frequent vomiting) and Varcobheda (diarrhoea). These are the Ekādaśarūpa of Rājayakṣmā caused by Vegasandhāraṇa (suppression of natural urges).

Kṣaya Yakṣmā Nidāna, Saṃprāpti and Lakṣaṇās

- ईर्ष्योत्कण्ठाभयत्रासक्रोधशोकातिकर्शनात्। अतिव्यवायानशनाच्छुक्रमोजश्च हीयते॥
ततः स्नेहक्षयाद्वायुर्वृद्धो दोषावुदीरयन्। प्रतिश्यायं ज्वरं कासमङ्गमर्दं शिरोरुजम्॥
श्वासं विड्भेदमरुचिं पार्श्वशूलं स्वरक्षयम्। करोति चांससन्तापमेकादशगदानिमान् ॥
लिङ्गान्यावेदयन्त्येतान्येकादश महागदम्। सम्प्राप्तं राजयक्ष्माणं क्षयात् प्राणक्षयप्रदम्॥ (Ca.Ci.8/24-27)

Atikarśana (excessive emaciation) caused by Īrṣyā (envy or greed), Utkaṇṭhā (anxiety), Bhaya (fear), Trāsa (frightening), Krodha (anger), Śoka (grief), Ativyavāya (excessive sexual intercourse) and Anaśana (fasting) leads to depletion of Śukra and Ojas (Rasa). Due to loss of these Sneha (unctuous substances), the Vāta gets aggravated which further aggravates Kapha and Pitta doṣas and produces Pratiśyāya (coryza), Jvara (fever), Kāsa (cough), Aṅgamarda (body ache), Śiroruja (headache), Śvāsa (dyspnoea), Varcobheda (diarrhoea), Aruci (anorexia), Pārśvaśūla (pain in the flanks), Svarakṣaya (aphonia) and Aṃsasantāpa (distress in the shoulders). These are the Ekādaśarūpa of Kṣayaja Rājayakṣmā which leads to Prāṇakṣaya (loss of life).

Viṣamāśana Yakṣmā Nidāna, Saṃprāpti and Lakṣaṇās

- विविधान्यन्नपानानि वैषम्येण समश्नतः। जनयन्त्यामयान् घोरान्विषमान्मारुतादयः॥
स्रोतांसि रुधिरादीनां वैषम्याद्विषमं गताः। रुद्ध्वा रोगाय कल्पन्ते पुष्यन्ति च न धातवः॥
प्रतिश्यायं प्रसेकं च कासं छर्दिमरोचकम्। ज्वरमंसाभितापं च छर्दनं रुधिरस्य च॥
पार्श्वशूलं शिरःशूलं स्वरभेदमथापि च। कफपित्तानिलकृतं लिङ्गं विद्याद्यथाक्रमम्॥ (Ca.Ci.8/28-31)

Due to Vividhānyannapānā (irregular intake of various food and drinks), the Vātādi doṣas aggravate and produces Ghorānviṣamān Āmayān (serious difficult disorders). Due to this irregularity in diet, the Vātādi doṣas having gone to disequilibrium, obstructs the Raktādi srotas and produces various disorders and improperly nourished dhātus which in turn produces the symptoms in sequential order viz Kapha, Pitta and Vāta. Due to vitiated Kapha, Pratiśyāya (coryza), Praseka (salivation), Kāsa (cough), Chardi (vomiting) and Arocaka (anorexia); due to vitiated Pitta, Jvara (fever), Aṃsasantāpa (distress in the shoulders) and Rudhirachardana (haematemesis) and due to vitiated Vāta, Pārśvaśūla (pain in the flanks), Śirośūla (headache) and Svarabheda (hoarseness of voice) are produced which are the Ekādaśarūpa of Viṣamāśanaja Rājayakṣmā.

Dhātu Kṣaya Śoṣa Lakṣaṇās

⊙ व्यवायशोकवार्धक्यव्यायामाध्वप्रशोषितान् । व्रणोरःक्षतसञ्ज्ञौ च शोषिणौ लक्षणैः शृणु ॥ (M.N..10/14)

⊙ व्यवायशोकस्थाविर्यव्यायामाध्वोपवासतः । व्रणोरःक्षतपीडाभ्यां शोषनन्ये वदन्ति हि ॥ (Su.U.41/16)

Ācārya Mādhavakara classified Śoṣa classified into seven types based on the Dhātu Kṣaya namely Vyavāya, Śoka, Vārdhakya, Vyāyāma, Adhva, Vraṇa and Uraḥkṣata. Whereas, Ācārya Suśruta classified Śoṣa classified into eight types namely, 1. Vyavāya, 2. Śoka, 3. Vārdhakya, 4. Vyāyāma, 5. Adhva, 6. Upavāsa, 7. Vraṇa and 8. Uraḥkṣata.

Vyavāya Śoṣa

⊙ व्यवायशोषी शुक्रस्य क्षयलिङ्गैरुपद्रुतः । पाण्डुदेहो यथापूर्वं क्षीयन्ते चास्य धातवः । (Su.U.41/17)

Śukra kṣaya liṅgarūpa (symptoms of deficiency of semen), Pāṇḍu dehā (pallor of the body) and Yathāpūrvaṃ kṣīyante cāsya dhātavaḥ (successive depletion of dhātus takes place in the inverse order) are the symptoms of Vyavāya Śoṣa (consumption due to excessive sexual intercourse).

Śoka Śoṣa

⊙ प्रध्यानशीलः स्रस्ताङ्गः शोकशोष्यपि तादृशः । विना शुक्रक्षयकृतैर्विकारैरभिलक्षितः । (Su.U.41/18)

Pradhyānaśīlaḥ (constant deep thought), Srastāṅga (looseness of the limbs) and Vinā śukrakṣayakṛtairvikārairabhilakṣitaḥ (present with all the clinical features of Vyavāya Śoṣa except the Śukra kṣaya (deficiency of semen)) are the symptoms of Śoka Śoṣa (consumption due to grief).

Vārdhakya Śoṣa

⊙ जराशोषी कृशो मन्दवीर्यबुद्धिवलेन्द्रियः । कम्पनोऽरुचिमान् भिन्नकांस्यपात्रहतस्वरः(नः) ॥
ष्ठीवति श्लेष्मणा हीनं गौरवारुचिपीडितः । सम्प्रस्रुतास्यनासाक्षः सुप्तरूक्षमलच्छविः ॥ (Su.U.41/19-20)

Kṛśā (emaciation), Mandavīryabuddhibalendriyaḥ (diminished virility, intellect, bodily strength and functions of sense organs), Kampa (tremors), Aruci (anorexia), Bhinnakāṃsyapātrahatasvaraḥ (his voice becomes like the sound of a cracked bell metal pot), Ṣṭhīvati śleṣmaṇā hīnaṃ (expectorations marked by absence of mucus), Gaurava (heaviness in the body), Aruci pīḍita (loss of taste), Samprasrutāsyanāsākṣaḥ (watery discharge comes out from his mouth, nose and eyes) and Suptarūkṣamalacchaviḥ (desiccated bodily wastes and dryness of the skin) are the symptoms of Vārdhakya Śoṣa (consumption due to old age).

Adhva Śoṣa

⊙ अध्वप्रशोषी स्रस्ताङ्गः सम्भृष्टपरुषच्छविः । प्रसुप्तगात्रावयवः शुष्कक्लोमगलाननः । (Su.U.41/21)

Srastāṅga (looseness of the limbs), Sambhṛṣṭa paruṣacchaviḥ (dried and rough skin), Prasuptagātrāvayavaḥ (numbness of the body parts) and Śuṣka klomagalānanaḥ (dryness of Kloma, throat and mouth) are the symptoms of Adhva Śoṣa (consumption due to excessive travelling).

Vyāyāma Śoṣa

⊙ व्यायामशोषी भूयिष्ठमेभिरेव समन्वितः । उरःक्षतकृतैर्लिङ्गैः संयुक्तश्च क्षताद्विना ॥। (Su.U.41/22)

The symptoms of Vyāyāma Śoṣa (consumption due to excessive physical exercise) are the same as above but with greater degree along with the symptoms of Uraḥkṣata (chest wound) without Kṣata (a wound).

Upavāsa Śoṣa

⊙ उपवासशोषिलक्षणं दोषधातुमलक्षयवृद्धिविज्ञानीयोक्तं 'तत्र वातलाहारसेविन' इत्यादिकं ज्ञातव्यम्॥ (Ḍa.Su.U.41/22)

⊙ तत्र पुनर्वातलाहारसेविनोऽतिव्यायामव्यवायाध्ययनभयशोकध्यानरात्रिजागरण पिपासाक्षुत्कषायाल्पाशन प्रभृतिभिरुपशोषितो रसधातुः शरीरमननुक्रामन्नल्पत्वान्न प्रीणाति, तस्मादतिकार्श्यं भवति; सोऽतिकृशः क्षुत्पिपासाशीतोष्णवातवर्षभारादानेष्वसहिष्णुर्वातरोग प्रायोऽल्पप्राणश्च क्रियासु भवति, श्वासकासशोषप्लीहोदराग्निसादगुल्मरक्तपित्तानामन्यतममासाद्य मरणमुपयाति, सर्व एव चास्य रोगा बलवन्तो भवन्त्यल्पप्राणत्वात् । (Su.Sū.15/33)

Ācārya Ḍalhaṇa has mentioned that the symptoms of Upavāsa Śoṣa are outlined in the chapter Doṣadhātumalakṣayavṛddhivijñānīya adhyāya. Indulging in the Upavāsa (fasting) for long time leads to Rasa dhātu śoṣaṇa (reduces the quantity of Rasa dhātu) in sequence reducing the nourishment to the body, thus making the person Atikārśya and produce the symptoms such as Kṣutpipāsaśītoṣṇavātavarṣa bhārādāneṣvasahiṣṇuta (intolerance to hunger, thirst, cold, hot, breeze, rain and weight-carrying) and Vātarogaprāyo (afflicted mostly with Vātaja disorders) and Alpaprāṇaśca kriyāsu bhavati (low vitality in all activities) are the features of Kārśya and in sequence, they may suffer from Śvāsa, Kāsa, Kṣaya, Plīha, Udara, Agnisāda, Gulma, Raktapitta and finally it leads to Marana (death). All diseases occurring in them become Balavanta (strong) due to Alpaprāṇatvāt (low vitality).

Vraṇa Śoṣa

⊙ रक्तक्षयाद्वेदनाभिस्तथैवाहारयन्त्रणात् । व्रणितस्य भवेच्छोषः स चासाध्यतमः स्मृतः ॥ (Su.U.41/23)

Raktakṣaya (loss of blood), Vedanā (pain) and Āhārayantraṇāt (restricted diet) are the symptoms of Vraṇa Śoṣa (consumption due to ulcer) which is Asādhya (incurable).

Uraḥkṣata Śoṣa

⊙ व्यायामभाराध्ययनैरभिघातातिमैथुनैः । कर्मणा चाप्युरस्येन वक्षो यस्य विदारितम् ।
तस्योरसि क्षते रक्तं पूयः श्लेष्मा च गच्छति ॥ कासमानश्छर्दयेच्च पीतरक्तासितारुणम् ।
सन्तप्तवक्षाः सोऽत्यर्थं दूयनात्परिताम्यति ॥ दुर्गन्धवदनोच्छ्वासो भिन्नवर्णस्वरो नरः ॥ (Su.U.41/24-27)

Vyāyāma (physical exercise), Bhāra (lifting up heavy weights), Adhyayana (excessive and loud reading), Abhighāta (injury), Atimaithuna (excessive sexual intercourse) and other acts injurious to the chest produce the disease Uraḥkṣata. Thereafter, Raktaṃ pūyaḥ śleṣmā ca gacchati (blood, pus and mucus are discharged from the chest lesions). Kāsamānaśchardayecca pītaraktāsitāruṇam (yellowish, bright red, black or reddish coloured blood is expectorated by bouts of coughing), Santaptavakṣāḥ (oppressed chest or breathing), Atyarthaṃ dūyanāt paritāmyati (faints due to the excessive pain), Durgandhavadanocchvāsa (foul smell is emitted from the mouth and in the breath) and Bhinnavarṇasvara (complexion and voice is altered) are the symptoms of Uraḥkṣata Śoṣa (consumption due to chest wound).

Pūrvarūpa of Rājayakṣmā

◉ पूर्वरूपं प्रतिश्यायो दौर्बल्यं दोषदर्शनम्। अदोषेष्वपि भावेषु काये बीभत्सदर्शनम्॥
घृणित्वमश्नतश्चापि बलमांसपरिक्षयः। स्त्रीमद्यमांसप्रियता प्रियता चावगुण्ठने॥
मक्षिकाघुणकेशानां तृणानां पतनानि च। प्रायोऽन्नपाने केशानां नखानां चाभिवर्धनम्॥
पतत्रिभिः पतङ्गैश्च श्वापदैश्चाभिधर्षणम्। स्वप्ने केशास्थिराशीनां भस्मनश्चाधिरोहणम्॥
जलाशयानां शैलानां वनानां ज्योतिषामपि। शुष्यतां क्षीयमाणानां पततां यच्च दर्शनम्॥
प्राग्रूपं बहुरूपस्य तज्ज्ञेयं राजयक्ष्मणः॥ (Ca.Ci.8/33-38)

Pratiśyāya (coryza), Daurbalyaṃ (debility), Doṣadarṣanam adoṣeṣvapi (finding faults in faultless things), Kāye bībhatsadarśana (hideous or abhorrent features in the body), Ghṛṇitvam (feeling of disgust), Aśnataścāpi balamāṃsaparikṣaya (loss of strength and muscles in spite of consuming adequate food), Strīmadyamāṃsa priyata (desire for women, wine and meat), Priyatā cāvaguṇṭhane (desire to be always covered by something), Makṣikāghuṇakeśānāmtṛṇānāṃ patanāni ca prāyoannapāne (often falling of insects, flies, hair and straws in food and drinks), Keśānāṃ nakhānāṃ cābhivardhanam (abnormal growing of hairs and nails), Patatribhiḥ etaṅgaiśca śvāpadaiścābhigharṣaṇam (striking by birds, wasps and animals), Svapne keśāsthirāśīnām bhasmanaścādhirohaṇam (dreaming of climbing heaps of hair, bones and ashes), Jalāśayānāṃ śailānāṃ vanānāṃ jyotiṣāmapi śuṣyatāṃ kṣiyamāṇānāṃ patatāṃ yacca darśanam (visualization of water reservoirs, mountains, forests which are already dried or getting dried and falling of stars) are the premonitory symptoms of various types of Rājayakṣmā.

Rūpa of Rājayakṣmā

Ekādaśarūpa Rājayakṣmā

◉ कासोंऽसतापो वैस्वर्यं ज्वरः पार्श्वशिरोरुजा। छर्दनं रक्तकफयोः श्वासवर्चोगदोऽरुचिः॥
रूपाण्येकादशैतानि यक्ष्मणः॥ (Ca.Ci.8/45-46)

Kāsa (cough), Aṃsasantāpa (distress in the shoulders), Vaisvarya (loss of voice), Jvara (fever), Pārśvaśūla (pain in the flanks), Śiroruja (headache), Chardanaṃ Raktakaphayoḥ (vomiting of blood and mucus), Śvāsa (dyspnoea), Varcobheda (diarrhoea) and Aruci (anorexia) are the clinical features of Ekādaśarūpa Rājayakṣmā

◉ स्वरभेदोऽनिलाच्छूलं संलोचश्चांसपार्श्वयोः । ज्वरो दाहोऽतिसारश्च पित्ताद्रक्तस्य चागमः ॥
शिरसः परिपूर्णत्वमभक्तच्छन्द एव च । कासः कण्ठस्य चोद्ध्वंसो विज्ञेयः कफकोपतः ॥ (Su.U.41/12-13)

Svarabheda (hoarseness of voice), Śūlaṃ (pain) and Saṃlocaścāṃsapārśvayo (contraction of shoulders and flanks) due to Vāta; Jvara (fever), Dāha (burning sensation), Atisāra (diarrhoea), Raktasya cāgamaḥ (haemoptysis) due to Pitta; Śirasaḥ paripūrṇatva (fullness in the head), Abhaktacchanda (aversion to food), Kāsa (cough) and Kaṇṭhasya coddhvaṃsa (loss of voice) due to Kapha, are the clinical features of Ekādaśarūpa Rājayakṣmā

◉ पीनसश्वासकासां समूर्द्धस्वररुजोऽरुचिः॥ ऊर्ध्वं, विड्भ्रंशसंशोषावधः, च्छर्दिश्च कोष्ठगे।
तिर्यक्स्थे पार्श्वरुद्दोषे, सन्धिगे भवति ज्वरः॥ रूपाण्येकादशैतानि जायन्ते राजयक्ष्मिणः। (A.H.Ni.5/13-15)

Pīnasa (nasal catarrh), Śvāsa (dyspnoea), Kāsa (cough), Aṃsaruja (pain in the shoulders), Mūrddha ruja (headache), Svara (change of voice), Aruci (anorexia) are the symptoms when the doṣas are localized in the upper parts of the body; Viḍbhraṃśasaṃśoṣa (either more elimination of faeces or constipation) occurs when the doṣas are localized in the lower parts of the body; Chardi (vomiting) occurs when the doṣas are localized in the Koṣṭha; Pārśvaruja (pain in the flanks) occurs when the doṣas are localized in the Tiryak gati (sidewards); Jvara (fever) occurs when the doṣas are localized in the Sandhi (joints) are the clinical features of Ekādaśarūpa Rājayakṣmā

Ṣaḍrūpa Rājayakṣmā

◉ ....षडिमानि वा। कासो ज्वरः पार्श्वशूलं स्वरवर्चोगदोऽरुचिः॥ (Ca.Ci.8/46)

Kāsa (cough), Jvara (fever), Pārśvaśūla (pain in the flanks), Svaragada (hoarseness of voice), Varcogada (diarrhoea) and Aruci (anorexia) are the clinical features of Ṣaḍrūpa Rājayakṣmā

◉ भक्तद्वेषो ज्वरः श्वासः कासः शोणितदर्शनम् । स्वरभेदश्च जायेत षड्रूपे राजयक्ष्मणि ॥ (Su.U.41/11)

Bhaktadveṣa (aversion to food), Jvara (fever), Śvāsa (dyspnoea), Kāsa (cough), Śoṇitadarśana (haemoptysis) and Svarabheda (hoarseness of voice) are the clinical features of Ṣaḍrūpa Rājayakṣmā

Trirūpa Rājayakṣmā

◉ त्रिभिर्वा पीडितं लिङ्गैर्ज्वरकासासृगामयैः ॥ (Su.U.41/15)

Jvara (fever), Kāsa (cough), and Asṛgāmaya (haemoptysis) are the clinical features of Trirūpa Rājayakṣmā.

Ācārya Caraka has not described Trirūpa separately. Cakrapāṅī while commenting on this, he mentioned Śṛṅgagrāhikāya (holding horn of anyone thus counting) any three features may be taken as Trirūpa Rājayakṣmā.

◉ अंसपार्श्वाभितापश्च सन्तापः करपादयोः। ज्वरः सर्वाङ्गगश्चेति लक्षणं राजयक्ष्मणः॥ (Ca.Ci.8/52)

But some scholars take Aṃsapārṣvābhitāpa (distress in the shoulders and flanks), Karapāda santāpa (burning sensation of palms and soles) and Jvara (fever) as the clinical features of Trirūpa Rājayakṣma.

Pratiśyāya

◉ घ्राणमूले स्थितः श्लेष्मा रुधिरं पित्तमेव वा। मारुताध्मातशिरसो मारुतं श्यायते प्रति॥
प्रतिश्यायस्ततो घोरो जायते देहकर्शनः। तस्य रूपं शिरःशूलं गौरवं घ्राणविप्लवः॥
ज्वरः कासः कफोत्क्लेशः स्वरभेदोऽरुचिः क्लमः। इन्द्रियाणामसामर्थ्यं यक्ष्मा चातः प्रजायते॥ (Ca.Ci.8/48-50)

The Kapha or Rakta or Pitta located at the base of the nose move towards head in the patient whose head is afflicted with Vāta and produces Ghora Pratiśyāya (severe coryza) which leads to Deha karśana (emaciation of the body). Śiraśūla (headache), Śirogaurava (heaviness in the head), Ghrāṇaviplavaḥ (loss of smell), Jvara (fever), Kāsa (cough), Kaphotkleśa (excessive mucous discharge), Svarabheda (hoarseness of voice), Aruci (anorexia), Klama (exhaustion), Indriyāṇāmasāmarthyaṃ (weakness of sense organs) and then Yakṣmā manifests.

Niṣṭhivaṇa

◉ पिच्छिलं वहलं विस्रं हरितं श्वेतपीतकम्। कासमानो रसं यक्ष्मी निष्ठीवति कफानुगम्॥ (Ca.Ci.8/51)

The person afflicted with Yakṣmā along with bouts of cough expectorates Picchila (slimy), Bahalaṃ (thick), Visraṃ (putrid) and Haritaṃ śvetapītakamrasaṃ (greenish or white-yellowish matter) associated with Kapha (phlegm).

Jvara

◉ अंसपार्श्वाभितापश्च सन्तापः करपादयोः। ज्वरः सर्वाङ्गश्चेति लक्षणं राजयक्ष्मणः॥ (Ca.Ci.8/52)

Aṃsapārṣvābitāpa (distress in the shoulders and flanks), Karapāda santāpa (burning sensation of palms and soles) and Sarvāṅga Jvara (generalized fever) are the characteristic features of Jvara in Yakṣmā.

Svarabheda

◉ वातात्पित्तात्कफाद्रक्तात् कासवेगात् सपीनसात्। स्वरभेदो भवेद्वाताद्रूक्षः क्षामश्चलः स्वरः॥
तालुकण्ठपरिप्लोषः पित्ताद्वक्तुमसूयते। कफाद्भेदो विवद्धश्च स्वरः खुरखुरायते ॥
सन्नो रक्तविवद्धत्वात् स्वरः कृच्छ्रात् प्रवर्तते। कासातिवेगात् कषणः पीनसात्कफवातिकः॥ (Ca.Ci.8/53-55)

Svarabheda (hoarseness of voice) is caused by Vāta, Pitta, Kapha and Rakta, Kāsa vega (bouts of coughing) and Pīnasa (nasal catarrh). The voice affected by Vāta becomes Rūkṣa (rough), Kṣāma (feeble) and Cala (unstable); if affected by Pitta, there will be Tālukaṇtha pariploṣaḥ (burning sensation of the palate and throat) and Vaktumasūyate (dislike to speak); if it is by Kapha, the voice becomes Bheda vibaddhaśca svara (break as if obstructed) along with Khurakhurāyate (rattling sound); if affected by Rakta, the voice becomes Sanna vibbadhatvāt (low due to obstruction) and comes out with difficulty; if its affected by Kāsātivega (frequent and forceful bouts and coughing) then it produces Kaṣaṇaḥ (traumatized throat) and if affected by Pīnasa, the voice resembles the features of Kapha and Vāta doṣa.

Pārśvaśūla

◉ पार्श्वशूलं त्वनियतं सङ्कोचायामलक्षणम्। (Ca.Ci.8/56)

Pārśvaśūla (pain in the flanks) is irregular and manifest during Saṅkoca (constriction) and Āyāma (expansion) of chest in Yakṣmā patient.

Śiraśūla

◉ शिरःशूलं ससन्तापं यक्ष्मिणः स्यात्सगौरवम्॥ (Ca.Ci.8/56)

In case of Yakṣmā, Śiraśūla (headache) is associated with Santāpaṃ (heat) and Gauravam (heaviness).

Raktaṣṭhivaṇa

◉ अभिसन्ने शरीरे तु यक्ष्मिणो विषमाशनात्। कण्ठात्प्रवर्तते रक्तं श्लेष्मा चोत्क्लिष्टसञ्चितः॥ (Ca.Ci.8/57)

When the Yakṣmā patient become emaciated due to Viṣamāśana (irregular diet), the aggravated and then accumulated Rakta and Śleṣmā (phlegm) comes out (expectorates) through the throat.

Rakta Vamathu (Śoṇita darśana)

◉ रक्तं विबद्धमार्गत्वान्मांसादीन्नानुपद्यते । आमाशयस्थमुत्क्लिष्टं बहुत्वात् कण्ठमेति च॥ (Ca.Ci.8/58)

Due to obstruction in the Raktavaha srotas, the Rakta (blood) does not reach the Māṃsādi dhātus and gets accumulated in the Āmāśaya. By this obstruction, the quantity of blood increases in abundance there and then comes out through the throat.

Śvāsa

◉ वातश्लेष्मविबद्धत्वादुरसः श्वासमृच्छति। (Ca.Ci.8/59)

Due to the obstruction of Vāta and Kapha in the Uras (chest), the Śvāsa (dyspnoea) manifests in the Yakṣmā patient.

Atisāra

◉ दोषैरुपहते चाग्नौ सपिच्छमतिसार्यते॥ (Ca.Ci.8/59)

Impairment of Agni (digestive fire) by the doṣas in the Yakṣmā patient results in Sapicchilātisāra (slimy loose stools).

Aruci

- ⊙ पृथग्दोषैः समस्तैर्वा जिह्वाहृदयसंश्रितैः। जायतेऽरुचिराहारे द्विष्टैरर्थैश्च मानसैः॥ कषायतिक्तमधुरैर्विद्यान्मुखरसैः क्रमात्। वाताद्यैररुचिं जातां मानसीं दोषदर्शनात्॥ (Ca.Ci.8/60-61)

Aruci (anorexia) is manifested when the doṣas individually or all together gets located in the Jihvā (tongue) and Hṛdaya (heart). And also Aruci manifests as Mānasika due to Dviṣṭairathai (disliked objects). Kaṣāya (astringent), Tikta (bitter) and Madhura (sweet) taste in the mouth help to understand the Aruci caused by Vāta, Pitta and Kapha respectively. Mānasika Aruci caused by Doṣa darśanāt (the sight of disliked objects).

Chardi

- ⊙ अरोचकात् कासवेगाद्दोषोत्क्लेशाद्भयादपि। छर्दिर्या सा विकाराणामन्येषामप्युपद्रवः॥ (Ca.Ci.8/62)

Chardi (vomiting) is caused by Arocaka (anorexia), Kāsavega (bouts of coughing) Doṣa utkleśa (aggravation of doṣas), Bhaya (fear) and also it occurs as an Upadrava (complication) of Anya vikāra (other diseases).

Upadrava of Rājayakṣmā

- ⊙ तेषामुपद्रवान् विद्यात्कण्ठोद्ध्वंसमुरोरुजम्॥ जृम्भाङ्गमर्दनिष्ठीववह्निसादास्यपूतिताः। (A.H.Ni.5/15-16)

Kaṇṭhoddhvaṃsa (abnormalities of throat), Uroruja (chest pain), Jṛmbha (yawning), Aṅgamarda (bodyache), Niṣṭhīva (expectoration of sputum), Agnisāda (weakness of digestive activity) and Āsyapūtitāḥ (bad smell of the mouth) are the complications of Rājayakṣmā.

Sādhyāsādhya of Rājayakṣmā

- ⊙ सर्वैरर्धैस्त्रिभिर्वाऽपि लिङ्गैर्मांसबलक्षये। युक्तो वर्ज्यश्चिकित्स्यस्तु सर्वरूपोऽप्यतोऽन्यथा॥ (Ca.Ci.8/47)

The person having Ekādaśarūpa, Ṣaḍrūpa or Trirūpa of Rājayakṣmā with Māṃsabala kṣaya (loss of muscles and strength) should be rejected or not treated (incurable). Otherwise, should be treated (curable) inspite of showing all the features of Rājayakṣmā.

- ⊙ महाशनं क्षीयमाणमतीसारनिपीडितम् । शूनमुष्कोदरं चैव यक्ष्मिणं परिवर्जयेत् ॥ उपाचरेदात्मवन्तं दीप्ताग्निमकृशं नवम् ॥ (Su.U.41/31-32)

Mahāśanaṃ kṣīyamāṇa (gradual wasting of the body in spite of intake of large amounts of food), Atisāranipīḍitam (suffering from diarrhoea) and Śūnamuṣkodaraṃ (swelling of the scrotum and abdomen) are the symptoms of a Yakṣmā patient who should be discarded from the treatment. Ātmavantaṃ (self-controlled), Dīptāgni (has good digestive power); Akṛśaṃ (not emaciated) and Navaṃ (recent origin of the disease) should be taken for the treatment.

In Contemporary science,

Rājayakṣmā is a broad spectrum disease which involves affliction of all srotas and in terms of Prāṇavaha Srotoduṣṭi, it can be correlated to pulmonary tuberculosis in contemporary science. (Please refer Topic 60).

MCQ's

1. Krodha is synonym of
a) Rājayakṣmā b) Uraḥkṣata
c) Kāsa d) Śvāsa

2. Which of the following is not a synonym of Rājayakṣmā
a) Yakṣmā b) Rogārāt
c) Jvara d) Uraḥkṣata

3. According to Nidāna, Rājayakṣmā is classified into _ types
a) 2 b) 4
c) 5 d) 6

4. According to Saṃprāpti, Rājayakṣmā is classified into .... types
a) 2 b) 4
c) 5 d) 6

5. According to Mādhavakara, Śoṣa is of .... types
a) 5 b) 6
c) 7 d) 8

6. सर्वधातुक्षयार्तस्य बलं तस्य हि विड्बलम् is the feature of
a) Rājayakṣmā b) Uraḥkṣata
c) Kāsa d) Śvāsa

7. पाण्डुदेह is seen in
a) Vyavāya Śoṣa b) Śoka Śoṣa
c) Vārdhakya Śoṣa d) Adhva Śoṣa

8. सम्प्रस्रुतास्यनासाक्षः is the feature of
a) Vyavāya Śoṣa b) Śoka Śoṣa
c) Vārdhakya Śoṣa d) Adhva Śoṣa

9. According to Suśruta, Trirūpa Rājayakṣmā are
   a) Jvara, Kāsa, Śvāsa
   b) Jvara, Śvāsa, Asṛgāmaya
   c) Jvara, Kāsa, Asṛgāmaya
   d) Aṃsapārśvābhitāpa, Karapāda santāpa and Jvara
10. According to Caraka, which of the following feature is not included in Ṣaḍrūpa Rājayakṣmā
   a) Varcogada b) Chardi
   c) Jvara d) Svaragada

Answer Keys

1	a	2	d	3	b	4	a	5	c
6	a	7	a	8	c	9	c	10	b

Short Answer Questions

1. Define Rājayakṣmā, Śoṣa, Kṣaya and Rogārāt
2. Explain the Nidāna and Saṃprāpti of Rājayakṣmā
3. Explain the types of Śoṣa
4. Explain the Pūrvarūpa of Rājayakṣmā
5. Explain Ekadaśarūpa Rājayakṣmā

◆

TOPIC 16

Common Lung Disorders

Introduction

Lung disorders, also known as respiratory or pulmonary diseases, impair the normal functioning of the lungs and affect millions of individuals globally. These conditions profoundly impact the quality of life, rendering everyday activities difficult. Lung disorders are typically classified into three primary categories:

- **Airway diseases :** These conditions affect the airways that carry oxygen and other gases in and out of the lungs. They often cause narrowing or blockage of the airways, which leads to obstructive lung diseases. Examples include asthma, chronic obstructive pulmonary disease (COPD), bronchiolitis, and bronchiectasis. People with airway diseases frequently describe the sensation of breathing out through a straw.
- **Lung tissue diseases :** These diseases affect the structure of the lung tissue. Inflammation or scarring makes it difficult for the lungs to expand fully, leading to restrictive lung disease. This restriction complicates the lung's ability to take in oxygen and release carbon dioxide. Individuals with this type of disorder often feel as though they are wearing a tight sweater or vest, making it hard to breathe deeply. Examples include pulmonary fibrosis and sarcoidosis.
- **Lung circulation diseases :** These diseases impact the blood vessels in the lungs, often caused by clotting, scarring, or inflammation. They hinder the lungs' ability to absorb oxygen and release carbon dioxide and may also affect heart function. An example of a lung circulation disease is pulmonary hypertension. People with these conditions often experience significant shortness of breath during physical exertion.

This chapter provides a comprehensive analysis of conditions such as Pneumonia, COPD, Pleural effusion and Bronchiectasis.

PNEUMONIA

Introduction

Pneumonia represents an inflammatory condition affecting the lungs, primarily characterized by the accumulation of fluid or pus within the alveoli, the tiny air sacs. This ailment can range from mild to severe, potentially fatal, affecting individuals across all age groups. Unfortunately, it stands as the most significant infectious cause of death among children on a global scale. As per data, India contributes approximately 23 per cent of the worldwide pneumonia burden and 36 per cent of the WHO regional burden among patients under five. There are no reliable estimates of disease burden, especially for adults.

Pneumonia can stem from various causative agents, encompassing bacteria, viruses, fungi, and parasites. Instances of pneumonia triggered by factors beyond pathogens, such as radiation, inhalation of toxic substances, allergic reactions, or pulmonary circulatory disorders, are infrequent. Infants, young children, individuals over 65 years of age, those with chronic co-morbid conditions, immunosuppressive disorders, and those who habitually smoke and have high alcohol intake are at a higher risk of contracting pneumonia.

Classification of Pneumonia: Pneumonia can be classified in several ways, including

- **Based on the etiology**
  - **Bacterial pneumonia** commonly stems from various bacterial pathogens, including Streptococcus pneumoniae, Staphylococcus aureus, Klebsiella pneumoniae, Haemophilus influenzae, Moraxella catarrhalis, anaerobes, and gram-negative organisms.
  - **Viral pneumonia** is attributed to viruses, with notable pathogens encompassing Respiratory syncytial virus (RSV), Rhinovirus, Influenza A, B and C viruses, adenoviruses
  - **Fungal pneumonia** arises from exposure to endemic fungi, such as Blastomyces, Coccidioides, and Histoplasma.
  - **Parasitic pneumonia** is rare but can occur. Examples include Strongyloides stercoralis and Toxoplasma gondii.
- **Based on the location of infection acquisition**
  - **Community-acquired pneumonia (CAP) :** Infection acquired within the community.
  - **Nosocomial Pneumonia**, which includes
    - **Hospital-acquired pneumonia (HAP) :** Infection acquired following a hospital stay of over 48 hours or in a patient recently discharged from the hospital (within 7 days).
    - **Ventilator-associated pneumonia (VAP) :** Infection acquired by a patient in the intensive care unit (ICU) who has been mechanically ventilated for over 48 hours.
    - **Healthcare-acquired pneumonia (HCAP) :** Infection acquired in healthcare facilities other than hospitals, such as nursing homes and dialysis centers.
- **Based on the severity of the infection**
  - **Mild Pneumonia** manifests as slight symptoms such as cough and fever and can be managed through rest and antibiotic therapy
  - **Moderate Pneumonia** leads to more pronounced symptoms, including breathing difficulties and chest pain, requiring prompt medical intervention for effective treatment.
  - **Severe Pneumonia** constitutes a medical emergency and may result in life-threatening complications.
- **Based on the area of lung affected**
  - **Lobar Pneumonia** refers to inflammation and consolidation that is confined to a specific lobe of the lung.
  - **Broncho pneumonia** refers to inflammation of the bronchi within the lungs.
  - **Multilobar pneumonia** is inflammation and consolidation of lung tissue affecting more than one lobe.

Pathophysiology

The pathophysiology of pneumonia begins with the entry of pathogens, such as bacteria, viruses, or fungi, into the lungs through inhalation, aspiration, or hematogenous spread. This triggers an immune response, activating immune cells and releasing cytokines and chemokines. The resulting inflammation and oedema impair gas exchange, disrupting surfactant production and causing alveolar collapse. Bronchial obstruction occurs due to inflammation and mucus production in bronchioles. Impaired gas exchange leads to hypoxemia and hypercapnia. Systemic effects include fever, chills, and inflammation, potentially progressing to sepsis, acute respiratory distress syndrome (ARDS), and multi-organ failure.

Clinical features:

Fever with chills, shortness of breath, chest pain during deep breaths, cough with sputum, haemoptysis, confusion, and signs of consolidation (expansion of the thorax on inspiration is reduced on the affected side, vocal fremitus is increased on the affected side, percussion note is impaired in the affected area, breath sounds are bronchial, possible medium, late, or paninspiratory crackles, vocal resonance is increased, pleural rub may be present and a lower PaO_2 than calculated in the alveolar gas equation).

Other symptoms may include a headache, muscle pain, extreme tiredness, nausea, vomiting and diarrhoea.

Complications:

In elderly patients and those with underlying health conditions, various complications may arise, including pleural effusion, empyema, lung abscess, acute respiratory distress syndrome, septicaemia, arrhythmia and multiple organ failure.

Diagnosis:

The diagnosis of pneumonia relies on a combination of clinical presentation, chest x-ray, blood cultures, sputum culture, urine antigen testing, and polymerase chain reaction (PCR). Clinical assessment encompasses tachypnea, tachycardia, diminished bronchial breath sounds, egophony, and tactile fremitus, all indicative of a consolidative process. Additional indicators include crackles upon auscultation of the affected lung regions and dullness upon percussion. Chest x-ray shows pulmonary infiltrate and helps identify the pattern of pneumonia. Computed tomography (CT) is a more reliable test and can be done when clinical symptoms suggest pneumonia despite a negative chest x-ray. Blood and sputum cultures help determine the specific micro-organism causing the pneumonia. Urine antigen tests identify antigens for Legionella and Streptococcus. Polymerase chain reaction (PCR) techniques can detect Legionella, Mycoplasma, and Chlamydia.

Prognostic scoring systems, such as CURB-65, prove to be effective in anticipating medical complications such as ICU admission and mortality. The CURB-65 pneumonia severity score is instrumental in categorizing patients based on specific criteria, including

- Confusion – 1 point
- Uremia (BUN greater than 20 mg/dL) – 1 point
- Respiratory rate exceeding 30 per minute – 1 point
- Hypotension (SBP less than 90 and DBP less than 60) – 1 point
- Age exceeding 65 years – 1 point

A CURB-65 score ranging from 0 to 1 suggests outpatient management, while a score of 2 or higher warrants hospitalization. Moreover, a score exceeding 4 calls for intensive care unit (ICU) admission and more rigorous therapeutic intervention.

BRONCHITIS

Introduction

Bronchitis is defined as the inflammation of bronchial tube in the lungs and it may be classified into acute bronchitis and chronic bronchitis. Acute bronchitis is an acute inflammation of bronchi which usually occurs following an upper respiratory infection, whereas chronic bronchitis occurs after prolonged or recurrent exposure to infectious or non-infectious irritation and it comes under the heading of COPD (Chronic Obstructive Pulmonary Diseases).

Acute bronchitis is the most common disease and is defined as lower respiratory tract infection causing an inflammation of the bronchi as a result of viral infections such as rhinovirus, influenza A or B virus, coronavirus, etc., or rarely it may be caused by bacterial infections such as Mycoplasma pneumoniae, Bordetella pertussis and Chlamydia pneumoniae or sometimes it may be caused by non-infectious inflammation such as exposure to irritants such as smokes, dusts, pollens or other irritants.

Pathophysiology:

The Pathophysiology of Acute bronchitis typically begins with an infection or irritation, most commonly caused by viral infections such as rhinovirus, influenza, and respiratory syncytial virus. Bacterial infections, allergens, and irritants like smoke and pollution can also trigger the condition. This infection or irritation leads to an inflammatory response in the bronchial walls, causing them to swell and produce excess mucus. The resulting inflammation causes mucosal thickening, epithelial-cell desquamation, and denudation of the basement membrane. These changes obstruct airflow and lead to a persistent cough, characterizing the primary symptom of acute bronchitis.

Clinical features:

The main symptom of acute bronchitis is cough and it lasts for 10 to 20 days but occasionally may last for 4 or more weeks and may become mucoid or mucopurulent in later stages. Other features are malaise, runny nose, sore throat, mild fever, chest discomfort, wheezing and shortness of breath.

Complications:

Usually, the acute bronchitis will not cause any severe complication as it gets cured if treated adequately. But in some cases, it leads to secondary pneumonia, chronic bronchitis and atelectasis of lung lobule.

Diagnosis:

Diagnosis is based on the clinical presentation and imaging studies. On clinical evaluation, tachycardia, tachypnoea may be present and on auscultation, vesicular breath sounds with prolonged expiration and wheezing may be heard. Rhonchi may be heard but it becomes clear with coughing and crepitations may be heard at the base of the lungs if the secretions are collected in the lungs. Imaging studies like chest x-ray helps to exclude the other diseases such as asthma, pneumonia, lung abscess, bronchiectasis and COPD.

CHRONIC OBSTRUCTIVE PULMONARY DISEASES (COPD)

Introduction

Chronic obstructive pulmonary disease (COPD) is defined as a disease state characterized by progressive development of chronic airflow limitation that is not fully reversible. Chronic obstructive pulmonary disease (COPD) is also known as chronic obstructive lung disease (COLD) and chronic obstructive airway disease (COAD).

In India, COPD is the second most common pulmonary disorder after tuberculosis and it is more prevalent in the middle aged male patients because of smoking habits. The most common causes of COPD's are cigarette smoke, exposure to poisonous particles or gases, alpha – 1 antitrypsin deficiency, hereditary and genetic factors resulting in airway obstruction or narrowing, hyperinflation of lungs and decreased lung elasticity. COPD includes chronic bronchitis and emphysema.

Chronic Bronchitis is clinically defined as chronic cough with expectoration for at least three months over two consecutive years. It is caused by chronic irritation from smoking and recurrent infections. Chronic bronchitis is classified into

- **Simple chronic bronchitis :** is characterized by productive cough but have no evidence of airflow obstruction.
- **Chronic mucopurulent bronchitis :** is characterized by persistent or recurrent purulent sputum production in the absence of localized suppurative disease such as Bronchiectasis.
- **Chronic obstructive bronchitis :** is characterized by long standing productive cough with severe dyspnoea due to airflow obstruction, usually accompanied by emphysema.

Emphysema is defined as the permanent abnormal enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of their walls without fibrosis. Emphysema is classified according to the pattern of involvement of acinus of the lung distal to the terminal bronchiole.

- **Centriacinar/Centrilobular emphysema** is characterized by destruction and enlargement of central or proximal part of respiratory bronchioles sparing distal air spaces. It is commonly seen in smokers in association with chronic bronchitis.
- **Panacinar/Panlobular emphysema** is characterized by uniform destruction and enlargement of acinus involved from respiratory bronchiole to terminal alveoli. It is associated with alpha -1 antitrypsin deficiency.
- **Paraseptal or Distal acinar emphysema** is characterized by destruction and enlargement of distal acinus next to a pleural surface.
- **Irregular emphysema** is characterized by scaring and damage of the acinus irregularly.

Pathophysiology:

The pathophysiology of COPD involves a combination of small airway disease (chronic bronchitis) and parenchymal destruction (emphysema), triggered by exposure to noxious particles and gases, most commonly from cigarette smoke. This exposure initiates an inflammatory response, leading to structural changes such as fibrosis and narrowing of airways, destruction of alveolar walls and loss of elastic recoil. These changes contribute to airflow limitation.

Additionally, inflammation increases mucus production, obstructing airways and reducing airflow. Over time, impaired gas exchange leads to symptoms like chronic cough, sputum production and shortness of breath. Reduced airflow and gas exchange contribute to hypoxemia (low oxygen levels) and hypercapnia (high carbon dioxide levels), exacerbating the condition and leading to systemic effects and comorbidities.

Clinical features:

The most common features of COPD are cough associated with sputum production and exertional dyspnoea. Although chronic bronchitis and emphysema are often co-existing, rarely the features of one type may be prominent.

In chronic bronchitis, the patient usually presents with a history of cough and sputum production for many years with a history of smoking. Initially the cough may be dry but subsequently it brings out sputum mucoid to mucopurulent and may be copious in amount. Over a period, the cough may occur in paroxysm or constant more in the early morning and it gets worsened in winter season. In advancing stage, exertional dyspnoea occurs due to airway obstruction and often complaints of chest tightness. The patient of chronic bronchitis is more prone to get infection and may develop fever and toxemia symptoms during an infection. Patients with chronic bronchitis are referred as Blue bloaters due to the presence of cyanosis and overweight.

In emphysema, the patient usually presents with a history of dyspnoea on exertion with minimal cough and production of mucoid sputum in small amount. The patient uses accessory respiratory muscles in the neck and abdomen; and has asthenic body with evidence of loss of weight. While sitting, the patient uses tripod position i.e. sits leaning forward and supporting the upper body with hands on the knees or on another surface and has tachypnoea with relatively prolonged expiration through pursed- lip breathing. The patients of emphysema are referred as Pink puffers due to maintenance of arterial oxygen tension (PaO_2).

In mild cases of COPD, clinical signs may be absent and in severe cases of COPD, the physical examination reveals signs of hyperinflation (increased anterio-posterior diameter leads to barrel-shaped chest), signs of hypercapnia (flapping tremor, bounding pulse, drowsiness, confusion and warm hands), signs of hypoxia (central cyanosis) are present. Clubbing is not a feature of COPD. Pitting edema may develop in patients with right heart failure, particularly if there is coexisting sleep apnoea or thromboembolic disease ('cor pulmonale') or renal hypoxia due to salt and water retention. Percussion note is hyper-resonant and cardiac and liver dullness is either absent or severely reduced.

On auscultation, patients with COPD may develop rhonchi and crepitations at the early stages of disease which change in the location and intensity after a deep and productive cough. In severe case of COPD, prolonged expiration, use of accessory respiratory muscles and the breath sounds are decreased

Complications:

Chronic Obstructive Pulmonary Disease (COPD) can lead to severe complications like pneumothorax (collapsed lung), respiratory failure, and chronic cor pulmonale (right-sided heart failure due to lung disease).

Diagnosis:

Diagnosis is based on the chest imaging studies and pulmonary function tests. Imaging studies like chest x-ray reveals bullae, increased translucency of lung fields, flattening of diaphragm, widened intercostal spaces and tubular heart. CT scan of chest reveals the extent and distribution of the emphysema and also suggest the co-morbidities such as pneumonia, lung cancer etc. Arterial blood gas analysis shows markedly reduced arterial oxygen (PaO_2) and elevated carbon dioxide ($PaCO_2$) levels.

Pulmonary function tests shows reduction of forced expiratory volume (FEV1) and forced expiratory volume/forced vital capacity (FEV1/ FVC) due to airflow obstruction. Peak expiratory flow rate (PEFR) is also reduced. With the severity of the disease, lung volumes may increase, resulting in an increase in total lung capacity (TLC), functional residual capacity (FRC) and residual volume (RV) in case of worsened disease.

PLEURAL EFFUSION

Introduction

Pleural effusion refers to the abnormal accumulation of fluid within the pleural space, while accumulations of pus (empyema) or blood (haemothorax) represent distinct conditions. An essential function of the pleural space is the generation and reabsorption of a minimal amount of fluid to facilitate pulmonary movement during respiration. However, pathological processes can disrupt this equilibrium, leading to excessive fluid retention. It is estimated that approximately 1.5 million individuals are affected by pleural effusion annually.

The presence of pleural fluid accumulation does not serve as a specific indication of a particular disease but rather as an indicative reflection of underlying pathology. Pleural effusions manifest in association with a diverse range of lung, pleural, and systemic disorders. According to Light's criteria, pleural effusion is categorized as either a transudate or exudate.

- **Transudative effusions** are caused by a combination of increased hydrostatic pressure and decreased plasma oncotic pressure. The most common causes are congestive cardiac failure, cirrhosis with ascites, and hypoalbuminemia due to nephrotic syndrome.
- **Exudative effusions** are caused by local processes that lead to increased capillary permeability, resulting in the exudation of fluid, protein, cells, and other serum constituents. Common causes include pneumonia, cancer, pulmonary embolism, viral infection, and tuberculosis. Light's criteria for exudative pleural effusion
  - Pleural fluid protein divided by serum protein > 0.5
  - Pleural fluid LDH divided by serum LDH > 0.6
  - Pleural fluid LDH more than two-thirds the upper limit of normal serum LDH

Pathophysiology

The pathophysiology of pleural effusion is characterized by an imbalance between the production and absorption of pleural fluid. This imbalance can be attributed to various disrupting factors, including:

- Elevated hydrostatic pressure, often associated with conditions such as heart failure.
- Reduced oncotic pressure, as seen in instances of hypoalbuminemia.
- Increased capillary permeability, which may occur due to infections, malignancies, or inflammatory processes.
- Impaired lymphatic drainage, commonly resulting from obstruction or malignancy.

The accumulation of excess fluid within the pleural space exerts compressive forces on the lung, thereby impairing its ability to expand. This leads to clinical manifestations that may include dyspnoea, chest pain, and a non-productive cough. Furthermore, inflammation of the pleura can provoke pleuritic chest pain, which typically intensifies during deep breathing or coughing.

Clinical features:

Pleural effusion may be asymptomatic, but possible symptoms include Pleuritic chest pain (a vague discomfort or sharp pain that worsens during inspiration, indicates inflammation of the parietal pleura, may be localized or referred), dyspnoea (the degree of which depends on the rate and the size of accumulation) and a dry, non-productive cough;

Diagnosis:

Diagnosis of pleural effusion is based on clinical examination, imaging studies, and pleural fluid analysis. Clinical examinations reveal tachypnoea, absent tactile fremitus, dullness to percussion, and decreased breath sounds on the side of the effusion, as well as tracheal and mediastinal shift to the opposite side. A pleural friction rub, while uncommon, is a classic physical sign detected best during inspiration and expiration over the left border of the sternum in the 3rd and 4th intercostal spaces.

Imaging studies, such as chest X-ray, serve as effective initial tools for identifying pleural effusion. The meniscus sign, observed in an upright posteroanterior (PA) view, signifies a substantial volume of pleural fluid, typically exceeding 200 ml, and blunts the costophrenic angle. Additionally, thoracic ultrasonography is capable of accurately detecting even small amounts of pleural effusion and is particularly useful in diagnosing loculated pleural effusion and for guiding thoracocentesis site marking. Computed tomography (CT) scanning can provide valuable insights into fluid characteristics, aiding in the diagnostic process.

Pleural fluid analysis involves the determination of protein, pH, lactate dehydrogenase, glucose, and albumin levels. In addition, adenosine deaminase levels and cell count are examined differentially and cytologically.

BRONCHIECTASIS

Introduction

Bronchiectasis is defined as persistent and irreversible dilatation of bronchi, which occurs due to congenital defects in mucociliary clearance, such as primary ciliary dyskinesia and cystic fibrosis. It may also arise from immune deficiencies, chronic inflammation, and recurrent infections within the airways. Globally, the prevalence of bronchiectasis varies between 4.2% and 21.2% across different populations. In India, the estimated prevalence

ranges from 2.3% to 6.4%, with incidence rates between 1.4 and 13.4 per 100,000 individuals per year. This condition is observed to be more prevalent among females in their fifth and sixth decades of life.

Bronchiectasis predominantly occurs in the left lower lobe, attributed to the longer and narrower anatomy of the lower lobe bronchus. Additionally, it may involve the lingula and the right middle lobe. Upper lobe involvement is relatively uncommon; however, it is often associated with conditions such as tuberculosis, cystic fibrosis, and allergic broncho-pulmonary aspergillosis. Bronchiectasis is classified morphologically into three distinct types:

- **Cylindrical or tubular Bronchiectasis:** This type is characterized by uniform bronchial dilation with smooth wall thickening.

- **Varicose Bronchiectasis:** In this classification, bronchial dilation is irregular and resembles varicose veins, frequently exhibiting a beaded appearance..

- **Saccular (cystic) Bronchiectasis:** This is the most severe form of bronchiectasis, wherein the bronchi are markedly dilated, terminating in dilated, and thick-walled cysts.

Pathophysiology

The pathophysiology of bronchiectasis is characterized by a cyclical process of infection and inflammation. An initial infection or airway insult results in damage to the airway walls, thereby impairing the clearance of mucus. This accumulation of mucus creates an environment conducive to bacterial colonization. The persistent presence of bacteria triggers chronic inflammation, which further the bronchial walls and cilia, the hair-like structures responsible for moving mucus out of the lungs. Over time, this damage becomes irreversible, leading to the distinct dilation of the airways. The continuous cycle of inflammation and infection sustains the condition, contributing to symptoms such as chronic cough, sputum production, recurrent respiratory infections, and dyspnoea.

Clinical features:

The clinical presentation commonly includes a persistent and recurrent cough, characterized by excessive sputum production, haemoptysis, pleuritic pain, and dyspnoea. Additional symptoms may encompass weight loss, fever, fatigue, malaise, anorexia, and halitosis (malodorous breath, often associated with purulent sputum). A physical examination may reveal the presence of nasal polyps and clubbing of fingers and toes. Auscultation of the lungs may reveal a combination of crackles, rhonchi, and wheezes.

Complications:

Complications associated with this condition may include chronic cor-pulmonale, pneumothorax, pyothorax, septic bronchopneumonia, amyloidosis, lung abscess and metastatic abscesses involving distant sites, such as the brain or pericardium.

Diagnosis:

Diagnosis is based on the sputum culture, chest imaging studies, bronchography and pulmonary function tests. Sputum cultures facilitate the identification of infectious organisms. Typically, the sputum exhibits a three-layered appearance: the upper layer is frothy and watery, the middle layer is turbid and mucopurulent, and the lower layer is purulent and opaque.

Imaging studies , including chest X-rays and CT scans, may demonstrate the signet ring sign (thickened, dilated airways appear adjacent to smaller artery resembling a signet (or pearl) ring, which is composed of a ring-shaped opacity in cross section), tram track sign (thickened non-tapering (parallel) walls of cylindrical bronchiectasis resembling the tramway tracks) and the gloved finger sign (mucoid impaction of the branching bronchi resembling the gloved finger).

Bronchography provides visual confirmation of bronchiectatic airways, while pulmonary function tests typically reveal a mixed pattern of obstructive and restrictive lung function.

MCQ's

1. Which causative agent is NOT commonly associated with pneumonia
a) Bacteria b) Viruses
c) Fungi d) Allergic reactions

2. What type of pneumonia is acquired in the community
a) Nosocomial Pneumonia
b) Healthcare-acquired Pneumonia (HCAP)
c) Ventilator-associated Pneumonia (VAP)
d) Community-acquired Pneumonia (CAP)

3. Which bacterial pathogen is commonly associated with bacterial pneumonia
a) Respiratory syncytial virus (RSV) b) Streptococcus pneumoniae
c) Adenoviruses d) Histoplasma

4. What is an effective prognostic scoring system for pneumonia severity
a) APACHE II b) CURB-65
c) SOFA d) MELD

5. What defines bronchitis
a) Inflammation of the alveoli
b) Inflammation of the bronchial tube in the lungs
c) Inflammation of the pleura
d) Inflammation of the trachea

6. What is the most common cause of acute bronchitis
a) Bacterial infections b) Viral infections
c) Fungal infections d) Parasitic infections

7. What complication can arise from untreated acute bronchitis
a) Heart failure b) Chronic bronchitis
c) Diabetes d) Liver cirrhosis

8. What is a typical duration for a cough associated with acute bronchitis
a) 2 to 5 days b) 10 to 20 days
c) 30 to 40 days d) More than 60 days

9. Which clinical sign might be heard on auscultation in acute bronchitis
a) Crackles at the lung base
b) Prolonged inspiration
c) Bronchial breath sounds
d) Vesicular breath sounds with prolonged expiration

10. How is COPD defined
a) A disease with fully reversible airflow limitation
b) A disease characterized by progressive development of chronic airflow limitation that is not fully reversible
c) An acute infection of the lungs
d) A genetic disorder affecting lung function

11. What is a common clinical feature of chronic bronchitis
a) Dry cough b) Productive cough with sputum
c) High fever d) Sharp chest pain

12. Which clinical feature is NOT typically associated with COPD
    - a) Hyperinflation
    - b) Clubbing
    - c) Cyanosis
    - d) Use of accessory respiratory muscles

13. What is pleural effusion
    - a) Inflammation of the alveoli
    - b) Abnormal accumulation of fluid within the pleural space
    - c) Accumulation of pus in the pleural space
    - d) Accumulation of blood in the pleural space

14. According to Light's criteria, how is pleural effusion categorized
    - a) Transudate and Exudate
    - b) Mild and Severe
    - c) Acute and Chronic
    - d) Infectious and Non-infectious

15. What is Bronchiectasis defined as
    - a) Reversible narrowing of bronchi
    - b) Persistent and irreversible dilatation of bronchi
    - c) Acute infection of bronchi
    - d) Temporary inflammation of bronchi

16. Which lobe is Bronchiectasis most commonly found in
    - a) Right upper lobe
    - b) Left lower lobe
    - c) Right lower lobe
    - d) Left upper lobe

17. What is a common clinical feature of Bronchiectasis
    - a) High fever
    - b) Persistent cough with excessive sputum production
    - c) Weight loss
    - d) Haemoptysis

18. What imaging sign is associated with Bronchiectasis on a chest X-ray or CT scan
    - a) Butterfly shadow
    - b) Signet ring sign
    - c) Ground glass opacity
    - d) Honeycombing

Answer Keys

1	d	2	d	3	b	4	b	5	b	6	b	7	b	8	b	9	d
10	b	11	b	12	b	13	b	14	a	15	b	16	b	17	b	18	b

Short Answer Questions

1. Define Pneumonia
2. Explain Bronchitis
3. Explain COPD
4. Explain Pleural effusion
5. Explain Bronchiectasis

◆

TOPIC 17

Jvara

Introduction

Jvara is considered as the foremost among all the diseases, because it severely afflicts the body, sense organs and mind along with diminution of intellect, strength, complexion, pleasure, enthusiasm and produces fatigue, lethargy, fainting and obstruction of food. It is associated at the time of birth and death in all living creatures as well. Jvara is the king of all diseases and destroyer of all creatures. Hence it should be treated very cautiously.

- ◉ रोगाणां रोगराजोऽयं यथा मृगपतिर्मृग। दाहात्मसु यथा वह्निस्तथा रोगो ज्वरऽधिकः।
  रुद्रक्रोधाग्निसम्भूतः सर्वभूतप्रतापनः॥ (Hā.Sa.Tṛ.2/31)

Further, Ācārya Hārīta described the predominance of Jvara with three similes, they are Rogānām rogarājoayam yathā mṛgapatirmṛga (as the lion adhers to highest status of animals, in the same way, the Jvara adhers the superior status as king among all the disease afflicting the human beings), Dāhātmasu yathā vahnistathā rogo jvaradhikaḥ (as the fire is strongest one among the inflammable things, in the same way, the Jvara is most powerful among all the diseases) and Rudrakrodhāgnisambhūtaḥ sarvabhūtapratāpanaḥ (since the Jvara is originated from anger of Lord Rudra and it is heating up extremely all the creatures).

And also he described the nomenclature of Jvara afflicting the animals; they are Nāganām (elephant) Pāka; Abhitāpastu Vājīnām (horse); Gavām (cow) Īśvara; Mānavānām (human being) Jvara; Dāridro Māhiṣa (buffalo); Mṛgarogo Mṛgeṣu (deer); Ajāvinām (goat and sheep) Pralāpa; Karabheṣva (camel) Alasā; Śuna (dog) Alarka; Matsya (fish) Indramata; Pakṣinām (birds) Abhighāta; Vyāla (snake) Aikṣita; Jālasya (water) Nīlika, Bhūmiṣu (land) Ūṣara and Vṛkṣa (tree) Koṭara. By these nomenclatures, we can understand that the Jvara afflicts all creatures of the universe (Jvaraḥ sarvatra dṛśyate).

Nirūkti and Paribhāṣā

- ◉ ज्वरयति शरीराणीति ज्वरः (Ca.Ni.1/35)

It is known as Jvara because it brings miseries to the body.

- ◉ ज्वरयति सन्तापयति। (Ca.Ca.Ni.1/35)

Since it increases the temperature of body, it is called Jvara.

- ◉ देहेन्द्रियमनस्तापी सर्वरोगाग्रजो बली। (Ca.Ci.3/4)

Jvara severely afflicts the body, sense organs and mind.

- ◉ स्वेदावरोधः सन्तापः सर्वाङ्गग्रहणं तथा ॥ विकारा युगपद्यस्मिन् ज्वरः स परिकीर्तितः । (Su.U.39/3)

The disease characterized by Svedāvarodha (absence of perspiration), Santāpa (rise of temperature) and Sarvāṅga graham (generalized body ache) collectively is known as Jvara.

Paryāya:

- ◉ ज्वरो विकारो रोगश्च व्याधिरातङ्क एव च। एकोऽर्थो नामपर्यायैर्विविधैरभिधीयते॥ (Ca.Ci.3/11)
- ◉ ज्वरो रोगपतिः पाप्मा मृत्युरोजोशनोऽन्तकः। क्रोधो दक्षाध्वरध्वंसी रुद्रोर्ध्वनयनोद्भवः॥ (A.H.Ni.2/1)

Jvara, Vikāra, Roga, Vyādhi, Ātaṅka, Rogapati, Pāpmā, Mṛtyu, Ojośana, Krodha, Dakṣādhvaradhvaṃsī and Rudrordhvanayanodbhava are synonymous and usually used to indicate this condition.

Jvara Prakruti:

- ◉ तस्य प्रकृतिरुद्दिष्टा दोषाः शारीरमानसाः। देहिनं न हि निर्दोषं ज्वरः समुपसेवते॥
  क्षयस्तमो ज्वरः पाप्मा मृत्युश्चोक्ता यमात्मकाः। पञ्चत्वप्रत्ययान्नृणां क्लिश्यतां स्वेन कर्मणा॥
  इत्यस्य प्रकृतिः प्रोक्ता, ...। (Ca.Ci.3/13-14)

The Śārīrika doṣas i.e. Vāta, Pitta and Kapha and the Mānasika doṣas i.e. Rajas and Tamas are the natural source of Jvara. The living being does not suffer from Jvara in absence of vitiation of Doṣas (Nirdoṣa).

Kṣaya (emaciation), Tama (feeling of entering into darkness), Jvara, Pāpmā (sinful acts) and Mṛtyu (death) all these are said to be the form of Yamā (God of death). As the god Yamā becomes causative agent for death or Pañcatva of those persons who are suffering due to their own deeds of the past life in the same way these diseases also kill the person due to deeds of past life.

Jvara Pravṛtti:

- ◉ ...प्रवृत्तिस्तु परिग्रहात्। निदाने पूर्वमुद्दिष्टा रुद्रकोपाच्च दारुणात्॥ (Ca.Ci.3/14)

The initial manifestation of Jvara is due to Parigrahā (tendency of greed or attachment). Earlier in the Nidāna Sthāna, it is described that the origin of Jvara is from the ferocious wrath of Rudra.

Jvara Prabhāva:

- सन्तापः सारुचिस्तृष्णा साङ्गमर्दो हृदि व्यथा। ज्वरप्रभावो, जन्मादौ निधने च महत्तमः। (Ca.Ci.3/26)

Santāpa (increased temperature), Aruci (anorexia), Tṛṣṇā (morbid thirst), Aṅgamarda (body ache), Hṛdi vyathā (pain in the cardiac region) are the effects of Jvara. During birth and death, Jvara is manifested in the form of Tamas (feeling as if entering the darkness).

Sāmānya Nidāna:

- मिथ्यातियुक्तैरपि च स्नेहाद्यैः कर्मभिर्नृणाम् ॥ विविधादभिघाताच्च रोगोत्थानात् प्रपाकतः ।
  श्रमात् क्षयादजीर्णाच्च विषात्सात्म्यर्तुपर्ययात् ॥ ओषधीपुष्पगन्धाच्च शोकान्नक्षत्रपीडया ।
  अभिचाराभिशापाभ्यां मनोभूताभिशङ्कया ॥ स्त्रीणामपप्रजातानां प्रजातानां तथाऽहितैः ।
  स्तन्यावतरणे चैव ज्वरो दोषैः प्रवर्तते ॥ (Su.U.39/19-22)
  - Mithyāti snehādi karma (excess or improper administration of oleation and allied therapies)
  - Vividhābhighātā (different kinds of injury)
  - Rogotthāna (manifestation of other diseases)
  - Prapāka (suppuration)
  - Śramāt (physical exertion)
  - Kṣaya (consumption)
  - Ajīrṇa (indigestion of food)
  - Viṣa (poison)
  - Sātmya ṛtu paryāyat (changes in habituations and seasons)
  - Auṣadhī puṣpagandha (inhalation of smell of plants and flowers)
  - Śoka (sorrow)
  - Nakṣatra pīḍayā (bad effects of stars and planets)
  - Abhicāra (effect of magical spells) or Abhiśāpa (curses),
  - Bhūtabhisaṅka (possession by evil spirits),
  - Strīṇāṃapa prajātānāṃ (improperly conducted labor in women)
  - Ahita prajātānām stanyāvataraṇe (improper conduct of women during the puerperal and lactation period).

Sāmānya Saṃprāpti:

- मिथ्याहारविहाराभ्यां दोषा ह्यामाशयाश्रयाः । बहिर्निरस्य कोष्ठाग्निं ज्वरदाः स्यू रसानुगाः ॥ (M.N.2/2)

The doṣās aggravated due to Mithyāhāra vihāra (improper food and behavior), enters the Āmāśaya and gets associated with Rasa; removes the Koṣṭhāgni from its place and this Agni pervade the entire body and produces Jvara.

- दोषाः प्रकुपिताः स्वेषु कालेषु स्वैः प्रकोपणैः ॥ व्याप्य देहमशेषेण ज्वरमापादयन्ति हि ।
  दुष्टाः स्वहेतुभिर्दोषाः प्राप्यामाशयमूष्मणा ॥ सहिता रसमागत्य रसस्वेदप्रवाहिणाम् ।
  स्रोतसां मार्गमावृत्य मन्दीकृत्य हुताशनम् ॥ निरस्य बहिरूष्माणं पक्तिस्थानाच्च केवलम् ।
  शरीरं समभिव्याप्य स्वकालेषु ज्वरागमम् ॥ जनयन्त्यथ वृद्धिं वा स्ववर्णं च त्वगादिषु । (Su.U.39/15-19)

The doṣās aggravated by their respective etiological factors pervade the entire body and thus produce Jvara. The aggravated doṣās enters into Āmāśaya and gets associated with Rasa dhātu along with heat, obstructs the passage of Rasa and Svedavahā srotas, causing Agnimāndhya and the Ūṣmaṇā (Agni) which is in Pakti sthāna (seat of digestion), pervade the entire body, initiating rise in temperature and exacerbations of Jvara during the seasonal variations of doṣic excitations and produces changes in the colour of the skin etc., based on the involvement of doṣas.

Saṃprāpti Ghaṭaka:

Doṣa	Pitta Pradhāna Tridoṣa
Dūṣya	Rasa and Sveda
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa and Sveda vaha
Srotoduṣṭi	Saṅga
Udbhava sthāna	Āmāśaya
Sañcāra sthāna	Rasavāhinyaḥ
Vyakta sthāna	Sarva deha
Rogamārga	Bāhya and Abhyantara

Sāmānya Pūrvarūpa

- आलस्यं नयने सास्रे जृम्भणं गौरवं क्रमः। ज्वलनातपवाय्वम्बुभक्तिद्वेषावनिश्चितौ॥
  अविपाकास्यवैरस्ये हानिश्च बलवर्णयोः। शीलवैकृतमल्पं च ज्वरलक्षणमग्रजम्॥ (Ca.Ci.3/28-29)
- श्रमोऽरतिर्विवर्णत्वं वैरस्यं नयनप्लवः । इच्छाद्वेषौ मुहुश्चापि शीतवातातपादिषु ॥
  जृम्भाऽङ्गमर्दो गुरुता रोमहर्षोऽरुचिस्तमः । अप्रहर्षश्च शीतं च भवत्युत्पत्स्यति ज्वरे ॥
  सामान्यतो, … । (Su.U.39/25-27)

Ālasya (laziness), Nayana srāva (lacrimation), Jṛmbhā (yawning), Gaurava (heaviness), Klama (fatigue), Bhakti and Dveṣa of Jvāla, Ātapa, Vāyu and Ambu (uncertainty about the liking and disliking for the heat, sun heat, wind and water); Avipāka (indigestion), Āsyavairasya (anorexia), Balavarṇahāni (loss of strength and complexion) and also Śīlavaikṛtamalpam (slight change in conduct) are the premonitory signs and symptoms of Jvara.

Viśeṣa Pūrvarūpa:

- …विशेषात्तु जृम्भाऽत्यर्थं समीरणात् । पित्तान्नयनयोर्दाहः, कफान्नान्नाभिनन्दनम् ॥ (Su.U.39/27)

Atyartha Jṛmbhā (excessive yawning), Nayana dāha (burning sensation of the eyes) and Annānnābhinandana (aversion to food) are the special premonitory symptoms of the derangement of Vāta, Pitta and Kapha Jvara respectively.

Classification of Jvara

- द्विविधो विधिभेदेन ज्वरः शारीरमानसः। पुनश्च द्विविधो दृष्टः सौम्यश्चाग्नेय एव वा॥
  अन्तर्वेगो वहिर्वेगो द्विविधः पुनरुच्यते। प्राकृतो वैकृतश्चैव साध्यश्चासाध्य एव च॥
  पुनः पञ्चविधो दृष्टो दोपकालवलावलात्। सन्ततः सततोऽन्येद्युस्तृतीयकचतुर्थकौ॥
  पुनराश्रयभेदेन धातूनां सप्तधा मतः। भिन्नः कारणभेदेन पुनरष्टविधो ज्वरः॥ (Ca.Ci.3/32-35)

Jvara is classified into two types:

- Śārīrika (physical) and Mānasika (mental)
- Saumya (predominated by cold) and Āgneya (predominated by heat)
- Antarvega (internal) and Bahirvega (external)
- Prākṛta (seasonal) and Vaikṛta (unseasonal)
- Sādhya (curable) and Asādhya (incurable)

It is again classified into five categories based on timings or period of strength and weakness of causative doṣas. They are Santata, Satata, Anyedyuṣka, Tṛtīyaka and Caturthaka. Again it is classified in to seven types based on the Āśraya (location) in the seven dhātus i.e. Rasa dhātugata, Rakta dhātugata, Māṃsa dhātugata, Medo dhātugata, Asthi dhātugata, Majjā dhātugata and Śukra dhātugata Jvara. As per the causes, the Jvara is classified into eight types; they are Vātaja, Pittaja, Kaphaja, Vātapittaja, Vātakaphaja, Pittakaphaja, Sannipātaja (Vātapittakaphaja) and Āgantuja Jvara.

Pratyātmaka Liṅga of Jvara:

- ज्वरप्रत्यात्मिकं लिङ्गं सन्तापो देहमानसः। (Ca.Ci.3/31)

The cardinal features of Jvara are Deha and Mana santāpa (feeling of excessive heat in the body and mind).

Śārīrika and Mānasika Jvara

- शारीरो जायते पूर्वं देहे, मनसि मानसः। वैचित्त्यमरतिर्ग्लानिर्मनसस्तापलक्षणम्॥ इन्द्रियाणां च वैकृत्यं ज्ञेयं सन्तापलक्षणम्। (Ca.Ci.3/36-37)

The Śārīrika jvara appears first in the body and the Mānasika jvara appears first in the mind. Vaicittya (mental distraction), Arati (disliking for everything) and Glāni (languor) are the signs and symptoms of the Mānasika tāpa (Jvara) and the functional abnormalities of the sense organs is the feature of Deha santāpa or Śārīrika jvara.

Saumya and Āgneya Jvara

- वातपित्तात्मकः शीतमुष्णं वातकफात्मकः॥ इच्छत्युभयमेतत्तु ज्वरो व्यामिश्रलक्षणः। (Ca.Ci.3/37-38)

The person seized by Vātapittaja jvara desires for cold articles, whereas if by Vātakaphaja jvara, he desires for hot articles, and in case of combined features, desires for both.

Antarvega and Bahirvega Jvara

- अन्तर्दाहोऽधिकस्तृष्णा प्रलापः श्वसनं भ्रमः॥ सन्ध्यस्थिशूलमस्वेदो दोषवर्चोविनिग्रहः।
  अन्तर्वेगस्य लिङ्गानि ज्वरस्यैतानि लक्षयेत्॥ सन्तापोऽभ्यधिको बाह्यस्तृष्णादीनां च मार्दवम्।
  बहिर्वेगस्य लिङ्गानि सुखसाध्यत्वमेव च॥ (Ca.Ci.3/39-41)

Antardāha (excessive burning sensation inside the body), Adhika tṛṣṇā (morbid thirst), Pralāpa (delirium), Śvasanaṃ (dyspnoea), Bhrama (giddiness), Sandhyasthi śūla (pain in bones and joints), Asveda (absence of sweating), Doṣavarcovinigraha (non elimination of doṣas and the feces) are the clinical features of Antarvega jvara. Santāpo abhyadhiko bāhya (excessive rise in the body temperature), Tṛṣṇā (mildness of thirst) and other features in mild form and easy curability are the features of Bahirvega jvara.

Prākṛta and Vaikṛta Jvara

- प्राकृतः सुखसाध्यस्तु वसन्तशरदुद्भवः। उष्णमुष्णेन संवृद्धं पित्तं शरदि कुप्यति॥
  चितः शीते कफश्चैवं वसन्ते समुदीर्यते। वर्षास्वम्लविपाकाभिरद्भिरोषधिभिस्तथा॥
  सञ्चितं पित्तमुद्रिक्तं शरद्यादित्यतेजसा। ज्वरं सञ्जनयत्याशु तस्य चानुबलः कफः॥
  प्रकृत्यैव विसर्गस्य तत्र नानशनाद्भयम्। अद्भिरोषधिभिश्चैव मधुराभिश्चितः कफः॥
  हेमन्ते, सूर्यसन्तप्तः सवसन्तेप्रकुप्यति। वसन्ते श्लेष्मणा तस्माज्ज्वरः समुपजायते॥ आदानमध्येतस्यापिवातपित्तं भवेद्नु।
  (Ca.Ci.3/42-47)

The Prākṛta jvara (natural or seasonal) manifesting in the Vasanta and Śarad seasons is easily curable. Pitta gets aggravated during Śarad (autumn) season because of its inherent hot property and hot environment. In the same way, the Kapha gets accumulated in Śita (cold) season and gets aggravated during Vasanta (spring) season.

The Pitta, getting accumulated during the rainy season due to the Amla vipāka of water and drugs gets aggravated during the autumn season and quickly produces Jvara. Kapha gets associated as subsidiary factor with Pitta. During this status, there is no harm by fasting because of the nature of Visarga kāla.

The Kapha, getting accumulated in Hemanta (early winter) season due to the sweetness of water and drugs gets aggravated during the Vasanta (spring) season due to its strong sunrays and produces Kaphaja jvara during the spring season which is the mid period of Ādana kāla. Vāta and Pitta also get associated as subsidiary factor with Kapha.

◉ कालप्रकृतिमुद्दिश्य निर्दिष्टः प्राकृतो ज्वरः॥ प्रायेणानिलजो दुःखः कालेष्वन्येषु वैकृतः। (Ca.Ci.3/48)

The Prākṛta jvara (natural or seasonal) is that which manifests according to nature of time period. However, Vātaja jvara irrespective of its season of manifestation, Pittaja jvara occurring in seasons except Śarad (autumn) season and Kaphaja jvara occurring in seasons except Vasanta (spring) season are said to be Vaikṛta jvara and it is difficult to cure.

Sādhya and Asādhya Jvara

◉ बलवत्स्वल्पदोषेषु ज्वरः साध्योऽनुपद्रवः। हेतुभिर्बहुभिर्जातो बलिभिर्बहुलक्षणः॥
ज्वरः प्राणान्तकृद्यश्च शीघ्रमिन्द्रियनाशनः। (Ca.Ci.3/50-51)

Jvara is easily curable (Sādhya) if it occurs in a Balavān (person of good strength), with Alpa doṣa (lesser amount of doṣas) and Anupadrava (absence of complications). Whereas Jvara caused by Bahu hetu (a strong etiological factor) associated with bahu lakṣaṇas (many clinical features) and Indriya nāśa (destroys the sense organs) are the characteristics of incurable types (Asādhya) of Jvara and it leads to death quickly.

◉ सप्ताहाद्वा दशाहाद्वा द्वादशाहात्तथैव च॥ सप्रलापभ्रमश्वासस्तीक्ष्णो हन्याज्ज्वरो नरम्।
ज्वरः क्षीणस्य शूनस्य गम्भीरो दैर्घरात्रिकः॥ असाध्यो बलवान् यश्च केशसीमन्तकृज्ज्वरः। (Ca.Ci.3/51-53)

Tīkṣṇa Jvara associated with Pralāpa (delirium), Bhrama (giddiness) and Śvāsa (dyspnoea) kills the patient either on the seventh, tenth or twelfth day. Gambhīra Jvara manifested in a Kṣīṇa (emaciated person) or Śūna (edematous person) and that occurring for very long durations (dīrgha rātrika) and causing a parting straight line in scalp hair (Keśasīmantakṛt) are incurable.

Sāma, Pacyamāna and Nirāma Jvara

◉ ज्वरोपद्रवतीक्ष्णत्वमग्लानिर्बहुमूत्रता। न प्रवृत्तिर्न विड्जीर्णा न क्षुत्सामज्वराकृतिः॥
ज्वरवेगोऽधिकं तृष्णा प्रलापः श्वसनं भ्रमः। मलप्रवृत्तिरुत्क्लेशः पच्यमानस्य लक्षणम्॥
जीर्णताऽऽमविपर्यासात्सप्तरात्रं च लङ्घनात्। (A.H.Ni.2/54-56)

◈ Tīkṣṇa upadrava (presence of complications in severe form), Aglāni (absence of debility), Bahu mūtrata (increased quantity of urine), Na pravṛttini viḍ (non elimination of feces), Na viḍ jīrṇeti (even if eliminated the faeces being not properly digested) and Na kṣut (absence of hunger) are the features of **Sāma Jvara.**

◈ Jvara vega adhikam (increase of body temperature), Tṛṣṇā (thirst), Pralāpa (delirium), Śvāsanaṃ (dyspnoea), Bhrama (dizziness), Mala pravṛtti (elimination of faeces) and Utkleśa (nausea) are the features of **Pacyamāna Jvara**

◈ Jīrṇa Jvara (fever becomes old due to lapse of time), appearance of features opposite of Āma Jvara such as presence of Kṣut (appetite), Kṣāmata (emaciation), Laghu gātrāta (lightness of the body), Jvaramārdhavam (mildness of fever) and Doṣa pravṛtti (elimination of doṣas);, fever after lapse of Sapta Rātram (seven days) and that after Laṅghana (fasting) are **Nirāma Jvara.**

Viṣama Jvara

◉ विषमो विषमारम्भक्रियाकालोऽनुषङ्गवान्। (A.H.Ni.2/69)

◉ ज्वरः पञ्चविधः प्रोक्तो मलकालबलाबलात्॥ प्रायशः सन्निपातेन भूयसा तूपदिश्यते।
सन्ततः सततोऽन्येद्युस्तृतीयकचतुर्थकौ॥ (A.H.Ni.2/56-57)

Viṣama Jvara is irregular in its Ārambha (onset), Kriyā (production of symptoms), Kāla (time of appearance) and possess Anuṣaṅga (persistence for long periods). It is classified into five categories based on strength and weakness of the doṣas. They are Santata, Satata, Anyedyuṣka, Tṛtīyaka and Caturthaka.

◉ दोषोऽल्पोऽहितसम्भूतो ज्वरोत्सृष्टस्य वा पुनः । धातुमन्यतमं प्राप्य करोति विषमज्वरम् ॥ (Su.U.39/66)

◉ केचिद्भूताभिषङ्गोत्थं ब्रुवते विषमज्वरम् ॥ (Su.U.39/68)

Indulgence in unwholesome diet and regimen by the convalescing person leads to aggravation of the residual doṣas, gets localized in one of the dhātus and produces Viṣama Jvara. Some scholars hold the view that Viṣama Jvara is due to Bhūtābhiṣaṅga (Grahāveśaḥ - possession by Grahās).

◉ सप्ताहं वा दशाहं वा द्वादशाहमथापि वा । सन्तत्या योऽविसर्गी स्यात्सन्ततः स निगद्यते ॥
अहोरात्रे सततको द्वौ कालावनुवर्तते । अन्येद्युष्कस्त्वहोरात्रादेककालं प्रवर्तते ॥
तृतीयकस्तृतीयेऽह्नि चतुर्थेऽह्नि चतुर्थकः । (Su.U.39/69-71)

◉ सततं रसरक्तस्थः सोऽन्येद्युः पिशिताश्रितः । मेदोगतस्तृतीयेऽह्नि त्वस्थिमज्जगतः पुनः ॥
कुर्याच्चातुर्थकं घोरमन्तकं रोगसङ्करम् । (Su.U.39/67-68)

◈ **Santata Jvara:** In this, the aggravated doṣas gets localized in Rasa dhātu and circulate through the Rasavaha srotas and spread all over the body and produces Santata Jvara. It also affects dhātus and Malas to manifest the diseases. The Santata Jvara is continuous for the period of 7 days, 10 days and 12 days according to predominant of Vāta, Pitta and Kapha respectively. During this period the Jvara may either subside or kill the patient.

- **Satata Jvara:** The aggravated doṣa being localized in the Rakta dhātu with presence of some counteracting factors produces Satata Jvara. It manifest twice within a day and night or twenty four hours.
- **Anyedyuṣka Jvara:** The aggravated doṣa with the support of any one factor amongst the Kāla, Prakṛti and Dūṣya in the presence of some counteracting factor obstruct the Medo vaha srotas and produces Anyedyuṣka Jvara, which rises once in day and night (24 hours).
- **Tṛtīyaka Jvara:** The aggravated doṣas gets localized in Asthi dhātu and manifest Jvara with the interval of one day. It manifests its effect in three ways viz. that manifesting due to Kapha and Pitta afflicts the Trikā (sacral) first; that due to Vāta and Kapha afflicts Pṛṣtha (back) first and that due to Vāta and Pitta afflicts the Śiras (head) first.
- **Caturthaka Jvara:** The aggravated doṣas gets localized in Majjā dhātu and manifest Jvara with the interval of two days. It manifests its effect into two ways viz. that which is caused by Kapha starts from Jaṅgha (calf region) and that which is caused by Vāta starts from Śiras (head).
- **Caturthaka Viparyaya Jvara** is a subtype of Caturthaka Jvara caused by three doṣas getting localized in two dhātus Asthi and Majjā dhātu. In this, the Jvara appears on 2nd and 3rd day whereas there will be absence of Jvara on 1st and 4th day.

Pralepaka Jvara

⊙ तथा प्रलेपको ज्ञेय:शोषिणां प्राणनाशनः । दुश्चिकित्स्यतमो मन्दः सुकष्टो धातुशोषकृत् ॥ (Su.U.39/54)

⊙ प्रलिम्पन्निव गात्राणि घर्मेण गौरवेण च । मन्दज्वरविलेपी च सशीतः स्यात्प्रलेपकः ॥ (M.N.2/41)

Pralepaka Jvara is characterised by Sakaṣṭa manda Jvara (low grade fever but very troublesome) and Dhātu śoṣa (loss of bodily elements) occurring in patients of Śoṣa (consumption), which is Prāṇa nāśana (fatal). Mādhavakara added some other lakṣaṇās that the Pralimpanniva gātrāṇi gharmeṇa (person feels as if the body smeared with heat), Gaurava (heaviness) and Saśīta (sensation of coldness).

Vātabalāsaka Jvara

⊙ नित्यं मन्दज्वरो रूक्षः शूनकस्तेन सीदति । स्तब्धाङ्गः श्लेष्मभूयिष्ठो भवेद्वातबलासकी ॥ (M.N.2/40)

Vātabalāsaka Jvara is characterized by Nithyaṃ manda Jvara (mild fever) occurring daily, Rūkṣa (dry), Śūnaka (swelling) and Stabdhāṅga (stiffness of the body) due to vitiation of Kapha doṣa.

Viparyaya Jvara

⊙ कफस्थानेषु वा दोषस्तिष्ठन् द्वित्रिचतुर्षु वा । विपर्ययाख्यान् कुरुते विषमान् कृच्छ्रसाधनान् ॥ (Su.U.39/55)

Viparyaya Jvara is caused by aggravated doṣas situated at the sites of Kapha and produces sudden and irregular attacks of Jvara on second, third or fourth day and is difficult to cure.

Dhātugata Jvara

⊙ गुरुत्वं दैन्यमुद्वेगः सदनं छर्द्यरोचकौ। रसस्थिते बहिस्तापः साङ्गमर्दो विजृम्भणम्॥
रक्तोष्णाः पिडकास्तृष्णा सरक्तं ष्ठीवनं मुहुः। दाहरागभ्रममदप्रलापा रक्तसंस्थिते॥
अन्तर्दाहः सतृण्मोहः सग्लानिः सृष्टविट्कता। दौर्गन्ध्यं गात्रविक्षेपो ज्वरे मांसस्थिते भवेत्॥
स्वेदस्तीव्रा पिपासा च प्रलापो वम्यभीक्ष्णशः। स्वगन्धस्यासहत्वं च मेदःस्थे ग्लान्यरोचकौ॥
विरेकवमने चोभे सास्थिभेदं प्रकूजनम्। विक्षेपणं च गात्राणां श्वासश्चास्थिगते ज्वरे॥
हिक्का श्वासस्तथा कासस्तमसश्चातिदर्शनम्। मर्मच्छेदो बहिः शैत्यं दाहोऽन्तश्चैव मज्जगे॥
शुक्रस्थानगतः शुक्रमोक्षं कृत्वा विनाश्य च। प्राणं वाय्वग्निसोमैश्च सार्धं गच्छत्यसौ विभुः॥
रसरक्ताश्रितः साध्यो मेदोमांसगतश्च यः। अस्थिमज्जगतः कृच्छ्रः शुक्रस्थो नैव सिद्ध्यति॥ (Ca.Ci.3/76-83)

- **Rasadhātugata Jvara:** Gurutva (heaviness), Dainya (miserable feeling), Udvega (anxiety), Sadana (exhaustion), Chardi (vomiting), Aruci (anorexia), Bahistāpa (external temperature), Aṅgamarda (body ache) and Jṛmbhā (yawning) are the features of Jvara located in Rasa dhātu.
- **Raktadhātugata Jvara:** Raktoṣṇa piḍakā (red and hot furuncles), Tṛṣṇā (thirst), Sarakta ṣṭīvanaṃ (hemoptysis), Dāha (burning sensation), Rāga (redness), Bhrama (giddiness), Mada (intoxication) and Pralāpa (delirium) are the features of Jvara located in Rakta dhātu.
- **Māṃsadhātugata Jvara:** Antardāha (burning sensation inside the body), Tṛṣṇā (thirst), Moha (unconsciousness), Saglāni (weakness), Sṛṣṭaviḍ (diarrhoea), Daurgandha (foul smell from the body) and Gātra vikṣepa (throwing movement of the limbs) are the features of Jvara located in Māṃsa dhātu.
- **Medodhātugata Jvara:** Tīvra sveda (excessive sweating), Pipāsā (thirst), Pralāpa (delirium), Vamyabhīkṣṇa (frequent vomiting), Svagandhasyāsahatva (inability to tolerate own odour), Glāni (laziness) and Aruci (anorexia) are the features of Jvara located in Meda dhātu.
- **Asthidhātugata Jvara:** Vireka (diarrhoea), Vamane (vomiting), Asthi bheda (tearing type pain in the bones), Prakūjana (gurgling sound in bowels), Gātrā vikṣepa (convulsive movements of the body parts) and Śvāsa (dyspnoea) are the features of Jvara located in Asthi dhātu.
- **Majjādhātugata Jvara:** Hikkā (hiccup), Śvāsa (dyspnoea), Kāsa (cough), Tamasaścātidarśana (feeling of entering into darkness frequently), Marmaccheda (cutting pain in the vital parts), Bahiḥ śaitya (feeling of coldness externally) and Antardāha (internal burning sensation) are the features of Jvara located in Majjā dhātu.

◈ **Śukradhātugata Jvara:** Aggravated doṣa causing Jvara located in the Śukra dhātu cause Śukra mokṣa (ejaculation) and Śukra vināśa (destruction of Śukra) is resulting in the extinction of life along with Vāyu, Agni and Soma of the subtle body or the person dies.

The Jvara located in the Rasa, Rakta, Māṃsa and Meda dhātu are easily curable; that located in the Asthi and Majjā dhātu are difficult to cure, and the Jvara located in Śukra dhātu is incurable.

Aṣṭavidha Jvara

◉ अथ खल्वष्टाभ्यः कारणेभ्यो ज्वरः सञ्जायते मनुष्याणां; तद्यथा- वातात्, पित्तात्, कफात्, वातपित्ताभ्यां, वातकफाभ्यां, पित्तकफाभ्यां, वातपित्तकफेभ्यः, आगन्तोरष्टमात् कारणात्॥ (Ca.Ni.1/17)

In human beings, Jvara manifest due to the eight causes such as Vāta, Pitta, Kapha, Vāta-pitta, Vāta-kapha, Pitta-kapha, Vāta-pitta-kapha and Āgantu (exogenous causes).

Vātaja Jvara:

◉ वेपथुर्विषमो वेगः कण्ठौष्ठपरिशोषणम् । निद्रानाशः क्षुतः स्तम्भो गात्राणां रौक्ष्यमेव च ॥
शिरोहृद्गात्ररुग्वक्त्रवैरस्यं बद्धविट्कता । जृम्भाऽऽध्मानं तथा शूलं भवत्यनिलजे ज्वरे ॥ (Su.U.39/29-30)

Vepathu (shivering), Viṣama vega (irregular fever), Kaṇṭhoṣṭapariśoṣanam (dryness of the throat, lips and of the mouth), Nidrā nāśa (loss of sleep), Kṣut stambha (stoppage of sneezing), Gātrāraukṣya (dryness of the body), Śiro hṛd gātra ruja (pain in the head, chest and body), Vaktra vairasya (distaste in the mouth), Viḍ baddha (constipation), Jṛmbhā (yawning), Ādhmāna (flatulence) and Śūla (abdominal pain) are the characteristics features of Vātaja Jvara.

Pittaja Jvara:

◉ वेगस्तीक्ष्णोऽतिसारश्च निद्राल्पत्वं तथा वमिः । कण्ठौष्ठमुखनासानां पाकः स्वेदश्च जायते ॥
प्रलापः कटुता वक्त्रे मूर्च्छा दाहो मदस्तृषा । पीतविण्मूत्रनेत्रत्वं पैत्तिके भ्रम एव च ॥ (Su.U.39/31-32)

Tīkṣṇa Vega (high fever), Atisāra (diarrhoea), Nidrā alpatva (disturbed sleep), Vami (vomiting), Kaṇṭhauṣṭa mukhanāsānāṃ pāka (inflammation in the throat, lips, mouth and nostrils), Sveda (perspiration), Pralāpa (delirium), Kaṭutā vaktra (pungent taste in the mouth), Mūrcchā (fainting), Dāha (burning sensation in the body), Mada (intoxication), Tṛṣa (thirst), Pīta viṇmūtranetra (yellowness of the stool, urine and eyes) and Bhrama (giddiness) are the symptoms of Pittaja Jvara.

Kaphaja Jvara:

◉ गौरवं शीतमुत्क्लेशो रोमहर्षोऽतिनिद्रता । स्रोतोरोधो रुगल्पत्वं प्रसेको मधुरास्यता ॥
नात्युष्णगात्रता छर्दिरङ्गसादोऽविपाकता । प्रतिश्यायोऽरुचिः कासः कफजेऽक्ष्णोश्च शुक्लता ॥ (Su.U.39/33-34)

Gaurava (heaviness), Śīta (feeling of cold), Utkleśa (nausea), Romaharṣa (horripilation), Ati nidratā (excessive sleep), Srotorodha (obstruction of the channels), Alpa ruja (mild pain), Prasekā (salivation), Madurāsyatā (sweet taste in the mouth), Nātyūṣṇa gātrata (slight heat in the body), Chardi (vomiting), Aṅgasada (lassitude), Avipāka (indigestion), Pratiśyāya (coryza), Aruci (anorexia), Kāsa (cough) and Akṣi śuklatā (whiteness of the eyes) are the symptoms of Kaphaja Jvara.

Vātapittaja Jvara:

◉ तृष्णा मूर्च्छा भ्रमो दाहः स्वप्ननाशः शिरोरुजा । कण्ठास्यदशोषो वमथू रोमहर्षोऽरुचिस्तथा ॥
पर्वभेदश्च जृम्भा च वातपित्तज्वराकृतिः । (Su.U.39/47-48)

Tṛṣṇā (thirst), Mūrcchā (fainting), Bhrama (giddiness), Dāha (burning sensation), Svapna nāśa (insomnia), Śirorujā (head ache), Kaṇṭhāsya śoṣa (dryness of throat and the mouth), Vamathu (vomiting), Romaharṣa (horripilation), Aruci (anorexia), Parvabheda (pain in the joints) and Jṛmbhā (yawning) are the characteristic features of Vātapittaja Jvara.

Vātakaphaja Jvara:

◉ स्तैमित्यं पर्वणां भेदो निद्रा गौरवमेव च ॥ शिरोग्रहः प्रतिश्यायः कासः स्वेदप्रवर्तनम् ।
सन्तापो मध्यवेगश्च वातश्लेष्मज्वराकृतिः ॥ (Su.U.39/49)

Staimitya (feeling of being covered with wet clothes), Parvaṇāṃ bheda (pain in the joints), Nidrā (sleepiness), Gaurava (heaviness of the body), Śirograhaḥ (head ache), Pratiśyāya (coryza), Kāsa (cough), Sveda pravartana (perspiration) and Madhya vega (moderate rise of temperature) are the characteristic features of Vātakaphaja Jvara.

Śleṣmapittaja Jvara:

◉ लिप्तातिक्तास्यता तन्द्रा मोहः कासोऽरुचिस्तृषा । मुहुर्दाहो मुहुः शीतं श्लेष्मपित्तज्वराकृतिः॥ (Su.U.39/50)

Liptāta (coating on the mouth), Tiktāsyatā (bitter taste in the mouth), Tandrā (drowsiness), Moha (unconsciousness), Kāsa (cough), Aruci (anorexia) Tṛṣā (thirst) and Muhurdāha muhurḥ śīta (alternate sensation of heat and cold) are the symptoms of Śleṣmapittaja Jvara.

Sannipātaja Jvara:

◉ निद्रानाशो भ्रमः श्वासस्तन्द्रा सुप्ताङ्गताऽरुचिः । तृष्णा मोहो मदः स्तम्भो दाहः शीतं हृदि व्यथा ॥
पक्तिश्चिरेण दोषाणामुन्मादः श्यावदन्तता । रसना परुषा कृष्णा सन्धिमूर्धास्थिजा रुजः ॥
निर्भुग्ने कलुषे नेत्रे कर्णौ शब्दरुगन्वितौ । प्रलापः स्रोतसां पाकः कूजनं चेतनाच्युतिः ॥
स्वेदमूत्रपुरीषाणामल्पशः सुचिरात् स्रुतिः । (Su.U.39/35-38)

Nidrā nāśa (insomnia), Bhrama (vertigo), Śvāsa (dyspnoea), Tandrā (drowsiness), Suptāṅgata (numbness in the limbs), Aruci (anorexia), Tṛṣṇā (thirst), Moha (unconsciousness), Mada (intoxication), Stambha (rigidity), Dāha (burning sensation) Śīta (feeling cold), Hṛdi vyathā (pain in the region of the heart), Pakticireṇa doṣa (delayed assimilation of the deranged bodily doṣas), Unmāda (insanity), Śyāva dantatā (blackish yellow discoloration on the teeth), Rasanā Paruṣā Kṛṣṇā (rough, dry and black tongue), Sandhi mūrdhāsthi ruja (pain in the joints, head and in the bone), Nirbhugne kaluṣe netra (sunken muddy eyes), Karṇau śabda ruja (pain and ringing in the ears), Pralāpa (delirium), Srotasāṃ pāka (inflammation of the channels), Kūjana (buzzing sound from the throat), Cetanācyuti (loss of consciousness) and Sveda mūtra purīṣāṇāmalpaśaḥ śucirāt sruti (scanty emission of perspiration, urine and fecal matter at long intervals) are the symptoms of Sannipātaja Jvara.

Ācārya Vāgbhaṭa mentioned Abhinyāsa Jvara and Hataujasa Jvara are the synonyms of Sannipātaja Jvara and is of 13 types, viz.

- **Vātapittolbaṇa mandakapha :** Bhrama (giddiness), Pipāsa (thirst), Dāha (burning sensation), Gaurava (heaviness) and Śiraso atiruk (excessive headache)
- **Vātakapholbaṇa mandapitta :** Śaitya (coldness), Kāsa (cough), Aruci (anorexia), Tandrā (drowsiness), Pipāsa (thirst), Dāha (burning sensation) and Vyathā (pain).
- **Pittakapholbaṇa mandavāta :** Chardi (vomiting), Śaitya muhur dāha (feeling of cold and burning sensation alternately), Tṛṣṇā (thirst), Moha (unconsciousness) and Asthi vedana (pain in the bones).
- **Vātolbaṇa mandapittakapha :** Sandhi asthi śūla (pain in the joints and bones), Śiro ruja (head ache), Pralāpa (delirium), Gaurava (heaviness), Bhrama (giddiness), Tṛṣṇā (thirst) and Kaṇṭhāsya śuṣkata (dryness of the throat and the mouth).
- **Pittolbaṇa mandavātakapha :** Rakta viṇmūtrata (blood in stool and urine), Dāha (burning sensation), Sveda (perspiration), Tṛṭ (thirst), Bala saṃkṣaya (loss of strength) and Mūrcchā (fainting).
- **Kapholbaṇa mandavātapitta :** Ālasya (laziness), Aruci (anorexia), Hṛllāsa (nausea), Dāha (burning sensation), Vamya (vomiting), Arati (uneasiness), Bhrama (giddiness), Tandrā (drowsiness) and Kāsa (cough).
- **Kapholbaṇa madhyapitta mandavāta :** Pratiśyāya (coryza), Chardi (vomiting), Ālasya (laziness), Tandrā (drowsiness), Aruci (anorexia) and Agni mārdavaṃ (weak digestion).
- **Pittolbaṇa madhyakapha mandavāta :** Hāridra mūtranetratvaṃ (yellowish discoloration of urine and eyes), Dāha (burning sensation), Tṛṣṇā (thirst), Bhrama (giddiness) and Aruci (anorexia).
- **Vātolbaṇa madhyakapha mandapitta :** Śiro ruja (headache), Vepathu (tremors), Śvāsa (dyspnoea), Pralāpa (delirium), Chardi (vomiting) and Aruci (anorexia).
- **Kapholbaṇa madhyavāta mandapitta :** Śīta (feeling of cold), Gaurava (heaviness), Tandrā (drowsiness), Pralāpa (delirium) and Asthi śiro atiruk (severe pain in bones and head).

- **Vātolbaṇa madhyapitta mandakapha :** Śvāsa (dyspnoea), Kāsa (cough), Pratiśyāya (coryza), Mukha śoṣa (dryness of the mouth) and Ati pārśva ruk (severe pain in the flanks).
- **Pittolbaṇa madhyavāta mandakapha :** Varco bheda (diarrhoea), Agni daurbalya (weak digestion), Tṛṣṇā (thirst), Dāha (burning sensation), Aruci (anorexia) and Bhrama (giddiness).
- **Tridoṣolbaṇa Sannipātaja Jvara :** Kṣaṇe dāha kṣaṇe śīta (alternate feeling of burning sensation and cold), Asthi sandhi śiro ruja (pain in the bones, joints and head), Netra srāva (excessive lacrimation), Kaluṣa rakta nirbhugna darśana (dirty, reddish and crooked eyes), Sasvanau saruja karṇa (tinnitus and pain in the ears), Kaṇṭha śukairivāvṛta (pricking pain in throat as if pricked by a thorn of grain), Tandrā (drowsiness), Moha (delusion), Pralāpa (delirium), Kāsa (cough), Śvāsa (dyspnoea), Aruci (anorexia) and Bhrama (giddiness), Paridagdhā khara sparśa jihvā (burnt tongue appearance and rough to touch), Sraṣṭāṅgata (excessive fatigue), Raktapittasya ṣṭīvanam (spitting of blood and bile mixed with phlegm), Śiraso loṭana (shaking of head), Tṛṣṇā (thirst), Nidra nāśa (insomnia) and Hṛdi vyathā (pain in the cardiac region), Svedamūtrapurīṣāṇāṃ cirāt alpa darśana (delayed and scanty discharge of sweat, urine and stool), Kṛśatvam nātigātrānāṃ (body part is not excessively emaciated), Pratataṃ kaṇṭhakūjanaṃ (constant cooing sound from throat), Koṭha (urticaria) and Śyava rakta maṇḍalā (whitish red circular patches appear on the skin), Mūkatva (aphonia), Srotasām pāka (inflammation of srotas), Gurutvamudarasya (heaviness of abdomen) and Cirāt pāka doṣāṇāṃ (delayed pāka of doṣas).

Ācārya Suśruta added Abhinyāsa, Hataujasa, Sanyāsa and Ojonirodhaja Jvara in Sannipātaja Jvara. And also he explained that Sannipātaja Jvara exacerbates again and then either subsides or becomes fatal on the seventh, tenth or the twelfth day.

Abhinyāsa Sannipātaja Jvara

- नात्युष्णशीतोऽल्पसञ्ज्ञो भ्रान्तप्रेक्षी हतस्वरः । खरजिह्वः शुष्ककण्ठः स्वेदविण्मूत्रवर्जितः ॥
  सास्रो निर्भुग्नहृदयो भक्तद्वेषी हतप्रभः । श्वसन्निपतितः शेते प्रलापोपद्रवायुतः ॥
  तमभिन्यासमित्याहुर्हतौजसमथापरे । सन्निपातज्वरं कृच्छ्रमसाध्यमपरे विदुः ॥ (Su.U.39/40-41)

Nātyuṣṇa śīta (neither very hot nor very cold temperature), Alpa saṃjñā (semi consciousness), Bhrāntapekṣī (confused look), Hata svaraḥ (lowered voice), Khara jihvā (rough tongue), Śuṣka kaṇṭha (dryness of throat), Sveda viṇmūtravarjita (non elimination of sweat, faeces and urine), Sāsrā (watering of the eyes), Nirbhugna hṛdaya (sinking heart), Bhakta dveṣī (aversion to food), Hata prabhaḥ (loss of lustre), Śvasannipatitaḥ śete (difficulty in breathing while lying down), Pralāpa (delirium) and Upadravāyutaḥ (presence of complications) are the features of Abhinyāsa sannipātaja jvara. Some Ācāryās call it is as Hataujasa jvara and it is considered as difficult to cure, while others opine that it is incurable.

Abhinyāsa, Hataujasa and Sanyāsa Sannipāta Jvara

- ⊙ निद्रोपेतमभिन्यासं क्षीणमेनं हतौजसम् । सन्न्यस्तगात्रं सन्न्यासं विद्यात्सर्वात्मके ज्वरे ॥ (Su.U.39/42)

If Sannipātaja Jvara is associated with Nidropetam (excessive sleep), it is known as Abhinyāsa Jvara, and if associated with Kṣīṇa (emaciation), it is known as Hataujasa Jvara and if associated with Sanyāsta gātra (complete flaccidity of body), it is called as Sanyāsa Jvara.

Ojonirodhaja Jvara

⊙ ओजो विस्रंसते यस्य पित्तानिलसमुच्छ्रयात् । स गात्रस्तम्भशीताभ्यां शयनेप्सुरचेतनः ॥
अपि जाग्रत् स्वपन् जन्तुस्तन्द्रालुश्च प्रलापवान् । संहृष्टरोमा स्रस्ताङ्गो मन्दसन्तापवेदनः ॥
ओजोनिरोधजं तस्य जानीयात् कुशलो भिषक् । (Su.U.39/43-45)

If Ojas is displaced due to aggravation of Pitta and Vāta, the person suffers from Gātra sthambha (stiffness of the body), Śītabhyo (coldness), Śayanepsuracetana (likes to sleep), Jāgrat svapan (loses consciousness often while awake or during sleep), Tandrā (feels drowsy), Pralāpa (delirium), Saṃhṛṣṭa roma (horripilation), Srasthāṅga (flaccidity of body parts), Manda Santāpa (mild fever) and Vedana (body pain). The expert physician should recognize this condition as Ojonirodhaja Jvara.

Ācārya Vāgbhaṭa added Dāhādi and Śītādi Sannipāta Jvara

⊙ अन्यच्च सन्निपातोत्थो यत्र पित्तं पृथक् स्थितम्। त्वचि कोष्ठेऽथवा दाहं विदधाति पुरोऽनु वा॥
तद्वद्वातकफौ शीतं, दाहादिर्दुस्तरस्तयोः। शीतादौ तत्र पित्तेन कफे स्यन्दितशोषिते॥
शीते शान्तेऽम्लको मूर्च्छा मदस्तृष्णा च जायते॥ (A.H.Ni.2/35-37)

When Pitta alone lodged in the Tvak (skin), it produces Dāha (burning sensation) at beginning. Whereas, it lodges in the Koṣṭha (alimentary tract) produces Dāha (burning sensation) at the end of the fever. When Vāta and Kapha lodged in the Tvak (skin), it produces Śīta (coldness) in the beginning. Whereas, in Koṣṭha (alimentary tract) produces Śīta (coldness) at the end of the fever. Among them, the Jvara which is associated with Dāha (burning sensation) at the beginning is difficult to cure.

In Śītādi sannipāta jvara, as Kapha gets liquefied in the Koṣṭha by aggravated Pitta, after Śīta subsides, there will be Amlaka (sour eructation), Mūrcchā (fainting), Mada (intoxication) and Tṛṣṇā (thirst). At the end of the Dāhādi sannipāta jvara, there will be Tandrā (stupor), Ṣṭīva (expectoration), Vami (vomiting) and Klamā (exhaustion).

Mādhavakara and Yogaratnākara explained 13 types of Sannipātaja jvara namely:

⊙ सन्धिकश्चान्तकश्चैव रुग्दाहश्चित्तविभ्रमः। शीतांगस्तन्द्रिकश्चैव कण्ड्गकुब्जश्च कर्णकः॥
विख्यातो भुग्ननेत्रश्च रक्तष्ठीवी प्रलापकः। जिह्वकश्चेत्यभिन्यासः सन्निपातास्त्रयोदश॥ (Y.R.2/27-28)

- ◈ Sandhika Sannipātaja jvara
- ◈ Āntaka Sannipātaja jvara

- ◈ Rugdāha Sannipātaja jvara
- ◈ Cittavibhrama Sannipātaja jvara
- ◈ Śītāṅga Sannipātaja jvara
- ◈ Tandrika Sannipātaja jvara
- ◈ Kaṇṭhakubja Sannipātaja jvara
- ◈ Karṇaka Sannipātaja jvara
- ◈ Bhugnanetra Sannipātaja jvara
- ◈ Raktaṣṭīvī Sannipātaja jvara
- ◈ Pralāpaka Sannipātaja jvara
- ◈ Jihvaka Sannipātaja jvara
- ◈ Abhinyāsa Sannipātaja jvara

Upadrava of Sannipātaja Jvara

- ⊙ सन्निपातज्वरस्यान्ते कर्णमूले सुदारुणः॥ शोथः सञ्जायते तेन कश्चिदेव प्रमुच्यते। (Ca.Ci.3/287-288)

At the end of Sannipātaja jvara, Karṇamūla śotha (severe swelling at the base (root) of ears) manifests and is incurable.

Asādhya Sannipātaja Jvara

- ⊙ दोषे विबद्धे नष्टेऽग्नौ सर्वसम्पूर्णलक्षणः॥ सन्निपातज्वरोऽसाध्यः कृच्छ्रसाध्यस्त्वतोऽन्यथा। (Ca.Ci.3/109-110)

Doṣe vibaddhe (Doṣas getting obstructed in the Srotas), Naṣṭe agni (digestive fire is completely destroyed) and Sarva sampūrṇa lakṣaṇas (manifestation of all clinical features), such Sannipāta jvara is incurable; otherwise it is curable with difficulty.

Āgantuja Jvara:

- ⊙ आगन्तुरष्टमो यस्तु स निर्दिष्टश्चतुर्विधः॥ अभिघाताभिषङ्गाभ्यामभिचाराभिशापतः। (Ca.Ci.3/111-112)

Āgantuja jvara is of four types, they are Abhighāta, Abhiṣaṅgaja, Abhicāra and Abhiśāpa.

Abhighātaja Jvara:

⊙ शस्त्रलोष्टकशाकाष्ठमुष्ट्यरत्नितलद्विजैः॥ तद्विधैश्च हते गात्रे ज्वरः स्यादभिघातजः।
तत्राभिघातजे वायुः प्रायो रक्तं प्रदूषयन्॥ सव्यथाशोफवैवर्ण्यं करोति सरुजं ज्वरम्। (Ca.Ci.3/112-114)

Jvara caused by the injury of body with weapons, stones, whip, wood, fist, slap, teeth and other such means is called Abhighātaja jvara. Here, due to the injury, the aggravated Vāta vitiates Rakta (blood) predominantly and produces Jvara associated with Vyathā (pain), Śopha (swelling), Vaivarṇa (discoloration) and Ruja (agony).

Abhiṣaṅgaja Jvara:

- कामशोकभयक्रोधैरभिषक्तस्य यो ज्वरः॥ सोऽभिषङ्गाज्वरो ज्ञेयो यश्च भूताभिषङ्गजः।
  कामशोकभयाद्वायुः, क्रोधात् पित्तं, त्रयो मलाः॥ (Ca.Ci.3/114-115)
- ग्रहावेशौषधिविषक्रोधाभिशोककामजः। अभिषङ्गात्..... ॥ (A.H.Ni.2/40)

Jvara caused by the affliction of Kāma (passion), Śoka (grief), Bhaya (fear), Krodha (anger) and Bhūta (evil spirits) is called Abhiṣaṅgaja jvara. Some opines Jvara caused by contact with Viṣa dravya and Viṣa vāyu also comes under Abhiṣaṅgaja jvara. Vāta gets aggravated by Kāma (passion), Śoka (grief) and Bhaya (fear), while Pitta is aggravated by Krodha (anger).

- ग्रहादौ सन्निपातस्य भयादौ मरुतस्त्रये। (A.H.Ni.2/43)

Vāgbhaṭa stated that all the three Doṣas are afflicted by Bhūta (evil spirits), Oṣadhigandha (odor of plants) and Viṣa (poison).

- ध्याननिःश्वासबहुलं लिङ्गं कामज्वरे स्मृतम्॥ शोकजे बाष्पबहुलं त्रासप्रायं भयज्वरे।
  क्रोधजे बहुसंरम्भं भूतावेशे त्वमानुषम्॥ मूर्च्छामोहमदग्लानिभूयिष्ठं विषसम्भवे। (Ca.Ci.3/122-124)
- ग्रहेणास्मिन्नकस्माद्धासरोदने॥ ओषधीगन्धजे मूर्च्छा शिरोरुग्वमथुः क्षवः। विषान्मूर्च्छातिसारास्यश्यावतादाहहृद्ग्रहाः। (A.H.Ni.2/40-41)
- श्यावास्यता विषकृते दाहातीसारहृद्ग्रहाः ॥ अभक्तरुक् पिपासा च तोदो मूर्च्छा बलक्षयः ।
  ओषधीगन्धजे मूर्च्छा शिरोरुक् वमथुः क्षवः ॥ कामजे चित्तविभ्रंशस्तन्द्राऽऽलस्यमरोचकः ।
  हृदये वेदना चास्य गात्रं च परिशुष्यति ॥ भयात् प्रलापः शोकाच्च भवेत् कोपाच्च वेपथुः ।
  अभिचाराभिशापाभ्यां मोहस्तृष्णा च जायते ॥ भूताभिषङ्गादुद्वेगहास्यकम्पनरोदनम् । (Su.U.39/76-80)

- The features of **Kāmaja jvara** are Dhyāna (constantly thinking same object) and Niśvāsa bahulam (excessive respiration).
- Baṣpa bahulam (excessive crying) in **Śokaja Jvara**
- Trāsaprāyam (threatened or terrorization) in **Bhayaja Jvara**
- Bahusaṃrambham (intense excitation) in **Krodhaja Jvara**
- Tvamānuṣam (superhuman behavior), Udvega (uneasiness), Hāsya (bursts of laughing), Kampa (tremors) and Rodhana (weeping) in **Bhūtaja jvara**
- Mūrcchā (fainting), Śiroruk (headache), Vamathu (vomiting) and Kṣavathu (sneezing) are the features of **Oṣadhigandhaja jvara** (fever due to inhalation of the odor of plants).
- Śyavasyata (bluish discoloration of mouth), Dāha (burning sensation), Atisāra (diarrhoea), Hṛdgraha (discomfort in the heart), Abhakta (aversion to food), Ruk (pain), Pipāsa (thirst), Toda (pricking pain), Bala kṣaya (loss of strength), Mūrcchā (fainting), Moha (unconsciousness), Mada (intoxication) and Glāni (fatigue) in **Viṣaja Jvara.**

Abhicāraja and Abhiśāpaja Jvara:

- अभिचाराभिशापाभ्यां सिद्धानां यः प्रवर्तते॥ सन्निपातज्वरो घोरः स विज्ञेयः सुदुःसहः।
  सन्निपातज्वरस्योक्तं लिङ्गं यत्तस्य तत् स्मृतम्॥ (Ca.Ci.3/118-119)
- तत्राभिचारिकैर्मन्त्रैर्हूयमानस्य तप्यते॥ पूर्वं चेतस्ततो देहस्ततो विस्फोटतृड्भ्रमैः।
  सदाहमूर्च्छैर्ग्रस्तस्य प्रत्यहं वर्द्धते ज्वरः॥ (A.H.Ni.2/44-45)

Abhicāra refers to enchantation of Mantras and Abhiśāpa refers to cursing. Abhicāraja and Abhiśāpaja jvara cause Sannipātaja jvara and it is an incurable condition. It produces the signs and symptoms which will be same that of Sannipātaja jvara.

In case of Abhicāraja jvara, the mind gets afflicted first and then the body and it produces features like Visphoṭa (eruptions), Tṛt (thirst), Bhrama (giddiness), Dāha (burning sensation), Mūrcchā (fainting) in the body and Pratyaham vardhete jvaraḥ (the Jvara increases day after day).

Jvara Mokṣa Lakṣaṇa

- ज्वरप्रमोक्षे पुरुषः कूजन् वमति चेष्टते। श्वसन्विवर्णः स्विन्नाङ्गो वेपते लीयते मुहुः॥
  प्रलपत्युष्णसर्वाङ्गः शीताङ्गश्च भवत्यपि। विसञ्ज्ञो ज्वरवेगार्तः सक्रोध इव वीक्ष्यते ॥
  सदोषशब्दं च शकृद्द्रवं स्रवति वेगवत्। लिङ्गान्येतानि जानीयाज्ज्वरमोक्षे विचक्षणः॥
  बहुदोषस्य बलवान् प्रायेणाभिनवो ज्वरः। सत्क्रियादोषपक्त्या चेद्विमुञ्चति सुदारुणम्॥
  कृत्वा दोषवशाद्वेगं क्रमादुपरमन्ति ये। तेषामदारुणो मोक्षो ज्वराणां चिरकारिणाम्॥ (Ca.Ci.3/324-328)

At the time of remission of Sannipātaja jvara, the patient develops Kūjana (groans), Vamati (vomiting), Ceṣṭate (becomes active), Śvasana (difficulty in breathing), Vivarṇa (discoloration), Svinnāṅga (perspiration of the body), Vepate (trembles), Līyate muhuḥ (frequent faints), Pralāpa (delirium), Atyūṣṇa sarvāṅga śītāṅga (body becomes very hot or very cold), Visamjña (unconsciousness), Jvara vegārta sakrodha (becomes angry during flare-up of Jvara) and Sadoṣa śabda sakṛddravaṃ sravati vegavat (forceful expulsion of liquid faeces with sound along with Doṣas).

In case of Abhinava (acute state) of Jvara with excessively aggravated Doṣas, with appropriate therapy and also maturation of Doṣas, there will be sudden remission (Dāruṇa mokṣa) of Jvara with difficulty. Whereas in the chronic state of Jvara, there will be gradual remission (Adāruṇa mokṣa) of Jvara without difficulty due to the withering of the vitiated Doṣas.

Jvara Mukti Lakṣaṇa

- विगतक्लमसन्तापमव्यथं विमलेन्द्रियम्। युक्तं प्रकृतिसत्त्वेन विद्यात् पुरुषमज्वरम्॥ (Ca.Ci.3/329)

Disappearance of Klama (exhaustion), Asantāpa (absence of temperature), Avyathaṃ (absence of discomfort or distress), Vimalendriya (clear senses) and Prakṛti sattvena (Sahaja satva-normalcy of mind) are the features of a person who has become free from Jvara.

Apathya during Jvara Mokṣa Kāla

- सज्वरो ज्वरमुक्तश्च विदाहीनि गुरूणि च। असात्म्यान्यन्नपानानि विरुद्धानि च वर्जयेत्॥
व्यवायमतिचेष्टाश्च स्नानमत्यशनानि च। तथा ज्वरः शमं याति प्रशान्तो जायते न च॥
व्यायामं च व्यवायं च स्नानं चङ्क्रमणानि च। ज्वरमुक्तो न सेवेत यावन्न बलवान् भवेत्॥ (Ca.Ci.3/330-332)

The person during Jvara, after remission of Jvara or during convalescence should not take food and drinks which are Vidāhī (producing burning sensation), Guru (heavy), Asātmya (unwholesome) and Viruddhā (mutually contradictory); and also one should avoid Vyavāya (sexual intercourse), Ati ceṣṭā (excessive activities), Snāna (bath) and Atyāśana (over eating). By following these rules, the fever gets alleviated and does not occur again. Vyāyāma (exercise), Vyavāya (sexual intercourse), Snāna (bath) and Caṅkramaṇa (walking) should be avoided by the person who has become free from Jvara, till he regains the strength.

Punarāvartaka Jvara

- असञ्जातबलो यस्तु ज्वरमुक्तो निषेवते। वर्ज्यमेतन्नरस्तस्य पुनरावर्तते ज्वरः॥
दुर्हृतेषु च दोषेषु यस्य वा विनिवर्तते। स्वल्पेनप्यपचारेण तस्य व्यावर्तते पुनः॥
चिरकालपरिक्लिष्टं दुर्बलं हीनतेजसम् ॥ अचिरेणैव कालेन स हन्ति पुनरागतः॥
अथवाऽपि परीपाकं धातुष्वेव क्रमान्मलाः। यान्ति ज्वरमकुर्वन्तस्ते तथाऽप्यपकुर्वते॥
दीनतां श्वयथुं ग्लानिं पाण्डुतां नान्नकामताम्। कण्डूरुक्तोठपिडकाः कुर्वन्त्यग्निं च ते मृदुम्॥
एवमन्येऽपि च गदा व्यावर्तन्ते पुनर्गताः। अनिर्घातेन दोषाणामल्पैरप्यहितैर्नृणाम्॥ (Ca.Ci.3/333-338)

The person, who has become free from Jvara, but before regaining the strength, if indulges in prohibited diet and mode of life, then the Jvara reappears. Even after becoming free from Jvara, but the Doṣas have not been eliminated completely, with Svalpenapyapacāreṇa (mild irregularities in diet and mode of life), the Jvara reappears. Patient who is suffering from Jvara for Cirakālaparikliṣṭa (long time), Durbala (is weak), Hīnatejas (has deficient luster), the relapsed fever leads to death quickly.

At times, Doṣas undergo Parīpāka in the Dhātus gradually and the fever subsides. But, their harmful effects continues and manifested as Dīnata (uneasiness), Śvayathu (edema), Glāni (lassitude), Pāṇḍuta (paleness), Nānna kāmatām (loss of appetite), Kaṇḍu (itching), Utkoṭha (urticaria), Piḍakā (pimples) and Mṛdu agni (subdued power of digestion). There occurs recurrence of other diseases which are already cured by the Doṣas being not eliminated properly, even by indulging in slight use of unwholesome regimens. Hence, even after complete remission of Jvara, depending upon the stage, strength and vitality of the Doṣas, they should be evacuated from the body either by elimination or alleviation therapies.

Jvara Upadrava

- श्वासो मूर्च्छाऽरुचिश्छर्दिस्तृष्णातीसारविड्ग्रहाः। हिक्का कासोऽङ्गभेदश्च ज्वरस्योपद्रवा दश॥ (Y.R.2/92)

Śvāsa (dyspnoea), Mūrcchā (fainting), Aruci (anorexia), Chardi (vomiting), Tṛṣṇā (thirst), Atisāra (diarrhoea), Viḍ grahā (constipation), Hikkā (hiccup), Kāsa (cough) and Aṅga bheda (breaking type of body pain) are the ten complications of Jvara.

In Contemporary sciecne,

Jvara can be correlated to Fever (pyrexia). (Please refer Topic 19)

MCQ's

1. According to Hārīta, Jvara is

a) रोगाणां रोगराजोऽयं यथा मृगपतिर्मृग
b) दाहात्मसु यथा वह्निस्तथा रोगो ज्वरऽधिकः
c) रुद्रक्रोधाग्निसम्भूतः सर्वभूतप्रतापनः
d) All of the above

2. Jvara in Matsya (fish) is known as

a) Pāka
b) Dāridro
c) Indramata
d) Ūṣara

3. Jvara in characterised by

a) Svedāvarodha
b) Santāpa
c) Sarvāṅga graham
d) All of the above

4. Doṣa Predominance in Jvara

a) Vāta
b) Pitta
c) Kapha
d) Rakta

5. Atyartha Jṛmbhā is the **Viśeṣa Pūrvarūpa of**

a) Vāta
b) Pitta
c) Kapha
d) Rakta

6. Pratyātmaka Liṅga of Jvara is

a) Deha Santāpa
b) Mana Santāpa
c) Dehamana Santāpa
d) None of the above

7. Doṣa Predominance in Saumya Jvara

a) Vāta
b) Kapha
c) Vātakapha
d) Vātapitta

8. In Satata Jvara, the doṣas gets localised in

a) Rasa dhātu
b) Rakta dhātu
c) Māṃsa dhātu
d) Asthi dhātu

9. What is the characteristic frequency of Anyedyuṣka Jvara

a) Occurs twice within 24 hour period
b) Occurs once in 24 hours
c) Occurs with a 48-hour interval
d) None of the above

10. Sakaṣṭa manda Jvara is the feature of

a) Vātabalāsaka Jvara
b) Viparyaya Jvara
c) Pralepaka Jvara
d) None of the above

11. According to Yogaratnākara, Sannipātaja jvara is of .... types

a) 8
b) 10
c) 11
d) 13

12. Karṇamūla śotha is the upadrava of

a) Vātaja Jvara
b) Pittaja Jvara
c) Kaphaja Jvara
d) Sannipātaja Jvara

13. Āgantuja Jvara is of .... types

a) 4
b) 7
c) 8
d) 13

14. ध्याननिःश्वासवहुलं is the feature of

a) Krodhaja Jvara
b) Kāmaja Jvara
c) Śokaja Jvara
d) Bhayaja Jvara

15. Lakṣaṇās of Oṣadhigandhaja jvara are

a) Mūrcchā, Śiroruk, Vamathu and Kṣavathu
b) Mūrcchā, Kṣavathu, Bāṣpa and Niśvāsa bahulam
c) Mada, Mūrcchā, Śiroruk and Vamathu
d) Mūrcchā, Atisāra, Glāni and Hṛdgraha

16. Jvara Upadrava are .... in number

a) 6
b) 8
c) 10
d) 12

Answer Keys

1	d	2	c	3	d	4	b	5	a	6	c	7	c	8	b
9	b	10	c	11	d	12	d	13	a	14	b	15	a	16	c

Short Answer Questions

1. Explain the classification of Jvara
2. Describe the characteristics of Sāma, Pacyamāna and Nirāma Jvara
3. Explain Viṣama Jvara
4. Describe Dhātugata Jvara
5. Explain Āgantuja Jvara
6. Explain Punarāvartaka Jvara

Long Answer Questions

1. Define Jvara and enlist the types of Jvara
2. Explain Nidāna, Saṃprāpti, Pūrvarupa, and Lakṣaṇās of Doṣaja Jvara
3. Describe Sannipātaja Jvara

◆

TOPIC 18

Masūrikā and Romāntika

Introduction

Masūrikā is one of the Kṣudraroga afflicting the body and face as a result of vitiation of Tridoṣa predominantly Pitta and Rakta. Ācārya Caraka, Suśruta and Vāgbhaṭa had described this disease in brief under the heading of Kṣudraroga. Mādhavakara had explained this disease in detail as a separate chapter.

⦿ भू-वायु-जलदोषादेर्भवन्त्यागन्तुहेतवः। संक्रामिणयो विशेषण निदाघादौ मघौ च ता॥ (Si.Ni.4/127)

Ācārya Gananāthasen in his Siddhānta Nidānam, described this disease under Āgantu Jvara (exogenous fever) because Viṣabhiṣaṅga (contact with poison) is the common cause of both the diseases. He also considered Masūrikā as Sankrāmiṇa (contagious) and Janapādika roga (epidemic disease) because this disease arises from the contamination of land, air, water etc., usually occurs during Nidāghā (summer) and Maghā (spring).

Paribhāṣā

⦿ मसूराकृतिः पिडका मसूरिका। लोके शीतलाशब्दवाच्या। (Āḍ.Śā.P.7/97-99)

The disease in which, there are Piḍakā (eruptions) resembling the shape of Masūra (lentil seeds) is called Masūrikā. Usually, it is expressed as Śītala in common language.

⦿ मसूराकृतिसंस्थानाः पिडकाः स्युर्मसूरिकाः । (M.N.54/3)

The disease in which, there are Piḍakā (eruptions) resembling the size and shape of Masūra (lentil seeds) is called Masūrikā.

Nidāna and Saṃprāpti

⦿ कट्वम्ललवणक्षारविरुद्धाध्यशनाशनैः । दुष्टनिष्पावशाकाद्यैः प्रदुष्टपवनोदकैः ॥ क्रूरग्रहेक्षणाच्चापि देशे दोषाः समुद्धताः । जनयन्ति शरीरेऽस्मिन् दुष्टरक्तेन सङ्गताः ॥ मसूराकृतिसंस्थानाः पिडकाः स्युर्मसूरिकाः । (M.N.54/1-3)

Intake of Kaṭvāmlalavaṇakṣāra (pungent, sour, saline and alkaline substances), Viruddhāśana (incompatible foods), Adhyāśanaśanai (taking food in excess quantity before the digestion of previous food), Duṣṭa niṣpāvaśākādyaiḥ (intake of contaminated beans, leafy vegetables), Praduṣṭa pavanodaka (ingestion of polluted air and water) and Krūragrahekṣaṇāccāpi deśe (influences of bad planetary on certain places in the country) are the causative factors that increases the doṣas in the body which is association with vitiated Rakta produces the Masūrākṛti saṃsthānāḥ piḍakā (eruptions resembling the size and shape of Masūra (lentil seeds).

⦿ पित्तं शोणितसंसृष्टं यदा दूषयति त्वचम्। तदा करोति पिडकाः सर्वगात्रेषु देहिनाम्। (Āḍ.Śā.P.7/97-99)

Ācārya Āḍhamalla stated that due to the causative factors the aggravated Pitta in association with Rakta vitiates the Tvak (skin) and produces Sarvagātreṣu dehinām piḍakā (eruptions all over the body).

Saṃprāpti Ghaṭaka:

Doṣa	Tridoṣa
Dūṣya	Rasa, Rakta, Māṃsa, Tvaca and Purīṣa
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa, Rakta and Māṃsa vaha
Srotoduṣṭi	Saṅga, Vimārga gamana
Udbhava sthāna	Āmapakvāśaya
Sañcāra sthāna	Tvaca
Vyakta sthāna	Tvaca
Rogamārga	Bāhya

Pūrvarūpa of Masūrikā

⦿ तासां पूर्वं ज्वरः कण्डूर्गात्रभङ्गोऽरतिर्भ्रमः ॥ त्वचि शोथः सवैवर्ण्यो नेत्ररागश्च जायते । (M.N.54/3-4)

Jvara (fever), Kaṇḍū (itching sensation), Gātrabhaṅga (pain in the body), Arati (restlessness), Bhrama (giddiness), Tvaciśotha (swelling), Vaivarṇyā (discoloration) and Netrarāga (redness of the eyes) are the premonitory symptoms of Masūrikā.

Masūrikā Bheda and Lakṣaṇās

◉ .....मसूरिका। चतुर्दशप्रकारेण् त्रिभिर्दोषैस्त्रिधा च सा। द्वन्द्वजा त्रिविधा प्रोक्तो सन्निपातेन सप्तमी॥
अष्टमी त्वग्गता ज्ञेया नवमी रक्तजा मता। दशमी मांसजा ख्याता चतदाोऽन्याश्च दुस्तरा॥
मेदोऽस्थिमज्जाशुक्रस्था: क्षुद्ररोगा इतीरिता:॥ (Āḍ.Śā.P.7/97-99)

Ācārya Śārṅgadhara classified Masūrikā into 14 types namely; one from each doṣa (Vātaja, Pittaja, Kaphaja), three from Dvandaja doṣas, seventh from Sannipātaja and eighth is Tvakgata, ninth Raktagata, tenth Māṃsagata and the remaining four are difficult to treat namely Medogata, Asthigata, Majjāgata and Śukragata Masūrikā.

Ācārya Mādhavakara classified it into 5 types Vātaja, Pittaja, Kaphaja, Raktaja and Sannipātaja Masūrikā and 7 types of Dhātugata Masūrikā.

Doṣaja Masūrikā Lakṣaṇās

Vātaja Masūrikā

◉ स्फोटा: श्यावारुणा रूक्षास्तीव्रवेदनयाऽन्विता: ॥ कठिनाश्चिरपाकाश्च भवन्त्यनिलसम्भवा: ।
सन्ध्यस्थिपर्वणां भेद: कास: कम्पोऽरति: क्लम: ॥ शोषस्ताल्वोष्ठजिह्वानां तृष्णा चारुचिसंयुता । (M.N.54/4-6)

The clinical features of Vātaja masūrikā are Śyāvāruṇa sphoṭā (dark brown or reddish blisters) which is associated with Rūkṣa (roughness), Tīvra vedana (severe pain), Kaṭhinā (hard), Cirapākā (suppurates slowly), Sandhyasthiparvaṇāṃ bheda (pain in the joints), Kāsa (cough), Kampa (tremors), Arati (restlessness), Klama (exhaustion), Talvoṣṭhaajihvā śoṣa (dryness of the palate, lips and tongue), Tṛṣṇā (thirst) and Aruci (anorexia).

Pittaja Masūrikā

◉ रक्ता: पीतासिता: स्फोटा: सदाहास्तीव्रवेदना: ॥ भवन्त्यचिरपाकाश्च पित्तकोपसमुद्भवा: ।
विड्भेदश्चाङ्गमर्दश्च दाहस्तृष्णाऽरुचिस्तथा ॥ मुखपाकोऽक्षिरागश्च ज्वरस्तीव्र: सुदारुण: । (M.N.54/6-8)

The clinical features of Pittaja Masūrikā are Raktāḥ pītaśītāḥ sphoṭāḥ (red, yellowish white blisters) which is associated with Sadāha (burning sensation), Tīvra vedanā (severe pain), Acirapākā (suppurates quickly), Viḍ bheda (diarrhoea), Aṅgamarda (body pain), Dāha (burning sensation), Tṛṣṇā (thirst) and Aruci (anorexia), Mukhapāka (mouth ulcers), Akṣi rāga (redness of the eyes), Tīvra jvara (high fever) and it is Sudāruṇa (dreadful).

Raktaja Masūrikā

◉ रक्तजायां भवन्त्येते विकारा: पित्तलक्षणा: ॥ (M.N.54/8)

The clinical features of Raktaja Masūrikā are similar to the features of Pittaja Masūrikā.

Kaphaja Masūrikā

◉ कफप्रसेक: स्तैमित्यं शिरोरुग् गात्रगौरवम् । हृल्लास: सारुचिर्निद्रा तन्द्रालस्यसमन्विता: ॥
श्वेता: स्निग्धा भृशं स्थूला: कण्डूरा मन्दवेदना: । मसूरिका: कफोत्थाश्च चिरपाका: प्रकीर्तिता: ॥ (M.N.54/8-10)

The clinical features of Kaphaja Masūrikā are Kapha praseka (salivation), Staimitya (as if the body covered with a wet cloth), Śiroruk (headache), Gātra gauravam (heaviness of the body) associated with Hṛllāsa (nausea), Aruci (anorexia), Nidrā (sleep), Tandrā (drowsiness), Ālasya (lassitude), Śveta piḍakā (whitish eruptions) which are Snigdha (soft), Bhṛśaṃ sthūla (extremely big in size), Kaṇḍū (itching sensation) and Mandavedanā (mild pain) and Cirapākā (suppurates slowly)

Sannipātaja Masūrikā

◉ नीलाश्चिपिटविस्तीर्णा मध्ये निम्ना महारुजा: । चिरपाका: पूतिस्रावा: प्रभूता: सर्वदोषजा: ॥
कण्ठरोधारुचिस्तम्भप्रलापारतिसंयुता: । दुश्चिकित्स्या: समुद्दिष्टा: पिडकाश्चर्मसञ्ज्ञिता: ॥ (M.N.54/11-12)

The clinical features of Sannipātaja Masūrikā are Nīla (bluish eruptions), Cipiṭa (flattened), Vistīrṇā (broad), Madhye nimnā (eruptions depressed in the centre), Mahāruja (severe pain), Cirapākā (suppurates slowly), Pūtisrāvā prabhūtāḥ (excessive putrid pus discharges); if associated with Kaṇṭharodha (obstruction in the throat), Aruci (anorexia), Stambha (stiffness of the body), Pralāpa (delirium) and Arati (restlessness) it is considered as Duścikitsā (difficult to cure). This condition is also known as **Carmapiḍakā or Carmadala.**

Dhātugata Masūrikā Lakṣaṇās

Tvakgata Masūrikā

◉ तोयबुद्बुदसङ्काशास्त्वग्गतास्तु मसूरिका: । स्वल्पदोषा: प्रजायन्ते भिन्नास्तोयं स्रवन्ति च ॥ (M.N.54/14)

The clinical features of Tvakgata masūrikā are Toyabudbuda saṅkaśā (eruptions resembling the bubbles of water) associated with Svalpa doṣa (mild increase of doṣa) and Bhinnastoyaṃ sravanti (thin watery discharge flows out when ruptured).

Raktagata Masūrikā

◉ रक्तस्था लोहिताकारा: शीघ्रपाकास्तनुत्वच: । साध्या नात्यर्थदुष्टाश्च भिन्ना रक्तं स्रवन्ति च ॥ (M.N.54/15)

The clinical features of Raktagata Masūrikā are Lohitākārā (reddish eruptions) associated with Śīgrapākā (suppurates quickly), Tanutvacaḥ (thin skin), Bhinnā raktaṃ sravanti (discharges blood when ruptured), Nātyarthaduṣṭā (no excessive increase of doṣas) and is Sādhya (curable).

Māṃsagata Masūrikā

◉ मांसस्था: कठिना: स्निग्धाश्चिरपाका घनत्वच: । गात्रशूलतृषाकण्डूज्वरारतिसमन्विता: ॥ (M.N.54/16)

The clinical features of Māṃsagata Masūrikā are Kaṭhinā snigdhā piḍakā (hard and unctuous eruptions), Acirapākā (suppurates slowly), Ghanatvacaḥ (thick skin) associated with Gātraśūla (body pain), Tṛṣṇā (thirst), Kaṇḍū (itching sensation), Jvara (fever) and Arati (restlessness).

Medogata Masūrikā

- ⊙ मेदोजा मण्डलाकारा मृदवः किञ्चिदुन्नताः । घोरज्वरपरीताश्च स्थूलाः स्निग्धाः सवेदनाः ॥
  सम्मोहारतिसन्तापाः कश्चिदाभ्यो विनिस्तरेत् । (M.N.54/17-18)

The clinical features of Medogat Masūrikā are Maṇḍalākārā mṛdava unnatā piḍakā (elevated, soft circular eruptions) associated with Ghorajvaraparītā (severe fever) and are Sthūla (large), Snigdha (unctuous), Savedanā (with pain), Saṃmoha (delusion), Arati (restlessness), Santāpa (distress) and someone recovers from this condition rarely.

Majjāgata Masūrikā

- ⊙ क्षुद्रा गात्रसमा रूक्षाश्चिपिटाः किञ्चिदुन्नताः ॥ मज्जोत्था भृशसम्मोहवेदनारतिसंयुताः । (M.N.54/18-19)

The clinical features of Majjāgata Masūrikā are Kṣudrā gātrasamā rūkṣā cipiṭā unnatā piḍakā (small, rough, flattened eruptions at the level of skin or slightly elevated) associated with Bhṛśa sammoha (severe delusion), Vedanā (painful) and Arati (restlessness).

Asthigata Masūrikā

- ⊙ छिन्दन्ति मर्मधामानि प्राणानाशु हरन्ति हि ॥ भ्रमरेणेव विद्धानि कुर्वन्त्यस्थीनि सर्वतः । (M.N.54/19-20)

The clinical features of Asthigata Masūrikā are Chindanti marmadhāmāni prāṇa nāśu haranti (cutting pain in the vital parts endangering life) associated with Bhramareṇeva viddhāni kurvantyasthīni sarvataḥ (pain in the bones as if stung by bee).

Śukragata Masūrikā

- ⊙ पक्वाभाः पिडकाः स्निग्धाः सूक्ष्माश्चात्यर्थवेदनाः ॥ स्तैमित्यारतिसम्मोहदाहोन्मादसमन्विताः ।
  शुक्रजायां मसूर्यां तु लक्षणानि भवन्ति हि ॥ निर्दिष्टं केवलं चिह्नं दृश्यते न तु जीवितम् । (M.N.54/20-22)

The clinical features of Śukragata Masūrikā are Snigdha sūkṣmā pakvābhā piḍakā (small unctuous ripened eruptions) which has Atyartha vedanā (severe pain) and associated with Staimitya (as if the body is covered with a wet cloth), Arati (restlessness), Sammoha (delusion), Dāha (burning sensation), Unmāda (insanity) and the patient may lose his life very soon.

- ⊙ दोषमिश्रास्तु सप्तैता द्रष्टव्या दोषलक्षणैः ॥ (M.N.54/22)

All these seven types of Masūrikā may be associated with vitiation of doṣas which are to be recognized as per the features of the doṣas present there.

BṚHAT MASŪRIKĀ, LAGHU MASŪRIKĀ AND ROMĀNTIKA

- ⊙ तत्राद्या बृहति नाम द्वितीया लघुनामिका। रोमान्तिका तृतीया च त्रिधा भिन्ना मसूरिका॥ (Si.Ni.4/126)

Ācārya Gananāthasen classified into three types namely, Bṛhat Masūrikā, Laghu Masūrikā and Romāntika.

Bṛhat Masūrikā

- ⊙ मसूरिकापिडकाः सान्द्रा गात्रेषु सर्वतः। भवन्ति पाकं गच्छन्ति लियन्ते च द्रुतं यतः॥
  नानोपद्रवसंयुक्तो ज्वरो यत्र सुदारुणः। बृहन्मसूरिका नाम शीतला चेति सा स्मृता॥(Si.Ni.4/129-130)

Bṛhat Masūrikā is characterized by Sāndra Piḍakā (dense eruptions) which resembles the Masūrikā (the size of shape of the lentils) which appears Sarvagātra (all over the body); gets Pāka gacchanti (suppurate in due course of time) and Liyante dṛtaṃ (subsides quickly) associated with Nānopdrava (different complications) and Dāruṇa Jvara (severe fever). It is also known as Śītala roga.

Saṃprāpti of Bṛhat Masūrikā

- ⊙ वाय्वादिदोषादथवान्यरोगिस्पर्शादथैतत् पिडकात्वचो वा। नासागलाद्यैः प्रविशद विषं तद्दोषानशेषान् विकरोति सद्यः॥
  ततोज्वरोःस्यादतिघोरलिङ्गोदोषाविषंतच्चबहिःक्षिपन्ति।गात्रेषुजाताःपिडकोस्ततस्ताःपक्वाःक्षयंयान्तिविषक्षयेणा॥
  तारतम्येन दोषाणां विषस्य च बलाबलात्। पृथग् वाऽत्यन्तसान्द्रा वा सरक्ता वा भवन्ति ताः॥ (Si.Ni.4/131-133)

The Viṣa (poison) present in the Vāyvādi etc., i.e. Bhū (land), Vāyu (air), Jala (water) enters into the body through Nāsāgalādya (nose and throat) and produces Piḍakātvaca (eruptions in the skin) or by Anya rogi sparśāt (touch of the diseased persons), the doṣas gets aggravated immediately and produces Ghoraliṅga Jvara (dreadful features of fever), then the aggravated doṣas push the Viṣa (poison) to the Bahiḥ (externally to the skin), and produces Gātreṣu jātāḥ piḍakā (eruptions all over the body). Pakvāḥ kṣayaṃ yānti viṣakṣayeṇā (when the eruptions gets suppurated, they disappears due to reduction in the poison). According to the strength of the doṣas and Viṣa (poison), the eruptions appears Pṛthak (separately) or Atyantasāndrā (profoundly together) or Saraktā (with blood).

Bṛhat Masūrikā Lakṣaṇās

- ⊙ आदितः शीतकम्पाभ्यां शिरःशूलेन च ज्वरः। प्रवर्त्तते वर्द्धते च कटीपृष्टं रुजन् भृशम्॥
  ततो मोहः प्रलापश्च प्रायेणान्येऽप्युपद्रवाः। तृतीये तु दिने प्राप्तो ज्वरो व्रजति लाघवम्॥
  पिडकाश्च प्रदृश्यन्ते गम्भीरास्त्वगधः स्थिताः। केशान्तुषु ललाटे च ताथाच मणिबन्धयोः॥
  समुद्भूय प्रसर्पन्ति मुखेऽङ्गषु च ताः क्रमात्। अत षष्ठे तु दिवसे प्रपूर्य्यन्ते जलेन ताः॥
  अष्टमेऽह्नि च पूयेन वर्द्धते च ज्वरस्ततः। शनैर्ह्रियते चोपि विषे प्रलघुतां गते॥
  प्रायशो द्वादशाहेन शुष्यन्ति पिडकाश्च ताः। विशर्य्यन्ते च पक्षेण त्रिसप्ताहैस्तु नैरुजम्॥

यावज्जीवं च लक्ष्मणि तिष्ठन्ति प्रायशस्त्वचि। विकाराह् सन्निपातोक्तास्तदन्तेभाविनश्च ये॥
ते प्रायेणात्र जायन्ते श्लेष्मबाधाश्च दारुणा:॥ (Si.Ni.4/134-141)

The clinical features of Bṛhat Masūrikā begins with Śīta (chills), Kampa (tremors), Śiraśūla (headache) and Jvara (fever), then the doṣas further increases and produce Kaṭipṛṣṭa ruja bhṛśam (severe pain in the waist and back), Moha (delusion), Pralāpa (delirium) and Anyopadravās (other complications).

On the third day, the Jvara vrajati lāghavaṃ (fever becomes mild) associated with Piḍakā pradṛśyante gambhīrāstvagadhaḥ sthitāḥ (manifestation of deep seated eruptions in the skin) appears on the Keśāntuṣu lalāṭe ca tāthāca maṇibandhayoḥ (border of the hairs on the forehead and wrists) and Sambudbhūya prasarpanti mukheaṅgeṣu ca tāḥ kramāt (gradually spreads into face and body).

On the sixth day, the eruptions get Prapūryyante jala (filled with water).

On the eighth day, there will be Pūyena varddhate (manifestation of pus inside the eruptions) along with Jvarastataḥ (appearance of fever) and the fever Śanakairhiyate cāpi viṣam pralaghutaṃ (gradually subsides due to reduction in the poison).

On the twelfth day, Śuṣyanti piḍakā (the eruptions becomes dry) and Viśaryyante ca pakṣeṇa (fall off from the skin).

After three weeks, Nairujam yāvajjīvam ca lakṣmaṇi tiṣṭhanti prāyaśastvaci (the disease disappears with the scar mark in the skin lifelong). Vikāraḥ sannipātaoktastadantebhāvinaśca ye te prāyeṇātra jāyante śleṣmabadhāśca dāruṇāḥ (even though the disease is caused by all the doṣas it has the predominance of Kapha doṣa).

Sādhyāsādhyata

◉ घोरे विषाभिषङ्गे तु दोषकोपात् सुदारुणात्। पिडका अतिसान्द्राः स्यु घोरश्चोपद्रवैज्वर:॥
अष्टाहात् परस्तेन प्रायश: स विपद्यते। कुथ्यन्ते क्वापि पिडका: ज्ञेया- तत्राप्यसाध्यता॥
शोणितेऽतिप्रदुष्टे तु रक्तपित्तं प्रवर्त्तते। रक्तपूर्णाश्च पिडका:, प्राणास्तत्रापि दुर्लभा:॥ (Si.Ni.4/141-143)

When the disease is caused by the Gore viṣābhiṣaṅge (dreadful poison), the doṣas get vitiated severely and produces Atisāndrā piḍakā (profoundly dense eruptions together) associated with Ghoraśca upadrava (dreadful complications) and Jvaraḥ (fever), then the patient dies after the period of eight days. Sometimes the eruptions become hard and Asādhya (incurable). When the Śoṇita atipraduṣṭe tu (blood becomes vitiated), it produces Raktapitta and Raktapūrṇā piḍakā (blood filled eruptions) and this condition leads to Praṇāstatrāpi durlabhāḥ (scarceness of life).

Laghu Masūrikā

◉ विरला: कतिचिद् यस्य पिडका: शीधूसम्भवा:। ज्वर: स्वल्पश्च तस्याऽसौज्ञेया लघुमसूरिका॥ (Si.Ni.4/148)

The disease manifested with Viralā kaṭicid yasya piḍakāḥ śīdhūsambhavāḥ (several small eruptions which appears quickly) and associated with Svalpa Jvara (mild fever) is known as Laghu Masūrikā.

Nidāna

◉ वाय्वादे रोगिसङ्गद् वा संक्रान्तं तद्विषं तनौ। दोषकोपं विधायाल्पं निरेति त्वचि सत्वरम्॥
ततस्ता: पिडका: स्वल्पा ज्वर: स्वल्पश्च जायते। शीघ्रमुल्लघुता चापि पिडकानां प्रशोषणात्॥ (Si.Ni.4/149-150)

Due to Vāyvāde (contamination of land, air, water etc.), Rogisaṅgad (touch of the patient), Saṅkrāntaṃ tadviṣaṃ (infection by poison), the doṣas get slightly aggravated and immediately moves to the skin and produces Svalpa piḍakā (small or few eruptions), Svalpa Jvara (mild fever) and Śīghramullaghutā cāpi piḍakānam praśoṣaṇāt (the person regains health quickly when the eruptions dry up).

Laghu Masūrikā Lakṣaṇas

◉ नातितीव्रो ज्वर: स्वल्पलिङ्ग: शीघ्रं प्रमुञ्चति। प्रथमेऽह्नि द्वितीये वा पिडका: सम्भवन्ति च॥
विरला: स्वल्पसंख्याका: क्षुद्रमौक्तिकसन्निभा:। अमध्यनिम्ना भिन्नस्तु तोयमात्रं स्रावन्ति ता:॥
पञ्चषैश्च दिनै: प्रायस्ता: प्रशुष्यन्ति सर्वत:। प्रायोऽष्टमेऽह्नि नैरुज्यं लक्ष्मणाञ्च द्रुतं लय:॥ (Si.Ni.4/151-154)

The clinical features of Laghu Masūrikā are Na atitīvra Jvara (mild fever), Svalpa liṅga (has few symptoms) and Śīghraṃ pramuñcati (liberates quickly).

On the first or second day, there will be appearance of Viralāḥ svalpasaṅkhyākāḥ Piḍakā (less number of scarcely eruptions) which resembles Kṣudramauktika sannibhāḥ (small pearls) with Amadhyanimnā (no depression in the center) and Bhinnastu toyamātraṃ sravanti (exude watery discharges when burst). On the fifth day, Praśuṣyanti sarvātaḥ (all eruptions gets dries up) and on the eighth day, Nairujyaṃ lakṣmaṇāñca drutaṃ layaḥ (the disease and its scar marks disappear).

Sādhyāsādhyata

◉ क्वचिद्विषस्य बाहुल्याद् दौर्बल्याद् काथ रोगिण:। कोथं गच्छन्ति पिडका: नानारूपा भवन्ति वा॥
रक्तेन वा प्रपूर्य्यन्ते ज्वरो घोरश्च जायते। कृच्छ्रसाध्यानसाधनं वा प्रकासांस्तान् प्रचक्षते॥ (Si.Ni.4/154-155)

Sometimes due to the Viṣaya bāhulyat (strength of the poison) or Daurbalyāt kātha rogiṇaḥ (weakness of the patient), the eruptions Kothaṃ gacchanti (gets putrefied) with Nānārūpa (different shapes) or Raktena prapūryyante (filled with blood) associated with Ghora Jvara (severe fever) and this condition is either Kṛcchra sādhya (difficult to cure) or Asādhya (incurable).

Romāntika

◉ रोमकूपोन्नतिसमा रागिण्य: कफपित्तजाः । कासारोचकसंयुक्ता रोमान्त्यो ज्वरपूर्विकाः ॥ (M.N.54/13)

Romāntika is characterized by Romakūponnati samā rāgiṇyaḥ (reddish eruptions elevated similar to that of hair follicles) as a result of aggravated Kapha and Pitta and is associated with Kāsa (cough), Arocaka (anorexia) and Jvarapūrvikā (preceded by fever).

⊙ निदानं पूर्ववत्तस्या विषं तु पृथगेव तत्। प्रायेणाभिभावेद् बालान् कफं पित्तञ्च कोपयेत्॥
श्वासमार्गादिषु प्रायो विकारान् व्रणशोथवत्। कुरुत्ते तत् क्रमात्, त्वक्ष निरस्तं च विलीयते॥
तात्रादितः प्रतिश्यायो ज्वरो रागश्च नेत्रयोः। तन्द्राऽरुचिः क्लमः कासो विड् भेदश्चापि कुत्रचित्॥
शोणायतानां ताल्वादौ दर्शन लक्ष्मणां ध्रुवम्॥ अथ कुर्य्यात्तृतीयाद्वा दिनात्ताम्राभलक्ष्मणाम्।
स्तोकोन्नतायतानां स्यान्मुखे गात्रेषु चोद्भवः॥ द्वित्रैश्च दिवसै स्तेषां भवेन्निःशेषनिर्गमः।
ततो ज्वरश्च लघुतां याति नो चेदुपद्रवाः॥त्र्यहाल्लयश्च चिह्नानां तुषाभत्वग्विमोक्षणात्। (Si.Ni.4/161-166)

Nidānaṃ pūrvavattasyā viṣaṃ tu pṛthageva tat (the cause of Romāntika is similar to Masūrikā with a difference in its toxin). Generally it affects children with aggravation of Kapha and Pitta and produces Śvāsamārgādiṣu prāyo vikārān (diseases of the respiratory channels) and Vraṇaśothavat kurutte tat kramā, tvakṣa nirastam ca vilīyate (the skin resembles like Vraṇa śotha (abscess) it gradually develops to subsequent stage and then disappear).

Romāntika begins with Pratiśyāya (coryza), Jvara (fever), Netrarāga (redness of the eyes), Tandrā (drowsiness), Aruci (anorexia), Klama (exhaustion), Kāsa (cough), Viḍbheda (diarrhoea) and Śoṇāyatānāṃ tālvādau darśana lakṣmaṇāṃ dhruvam (reddish eruptions or marks appears in the palate quickly). On the second or third day, the eruptions or marks become Tāmrābha lakṣmaṇāṃ (coppery red) and gradually it starts to appear on the Mukhegātreṣu udbhavaḥ (face and body). In another two or three days, the eruptions becomes Niḥśeṣa nirgama (vanished completely) associated with Jvaraśca laghutāṃ (the fever becomes mild) and Na upadravā (without complications). After three or more days, there will be Tuṣābhatvagvimokṣaṇāt (casting away of scales of skin resembling husk).

Sādhyāsādhyata of Romāntika

⊙ अथ चेद् रोगमध्ये वा रोगशेषेऽथवा क्वचित्। तद्विषं श्वासनलिकाः फुस्फुसौ च प्रसर्पति॥
तदा श्वासश्च कासश्च ज्वरवेगश्च वर्द्धते। मोहस्तन्द्रावसादश्च प्रायश्च प्राणसंशयः॥
म्रियन्ते च ततो बाला विशेषण सहदाशः। जीवन्त्युपक्रमात् केचित् सुचिरं दुर्बलीकृताः॥
गम्भीररक्तचिह्नानां रक्तपित्तस्य चेक्षणात्। वृते रक्तातिसारे वा रोमान्त्यां दुर्लभं सुखम्॥ (Si.Ni.4/167-170)

During the middle or end stage of the disease, the Viṣa (poison) enters the Śvāsanalikāḥ phusphusau (respiratory tract and spreads to the lungs), produces Śvāsa (dyspnoea), Kāsa (cough), Jvaravega vardhate (increase of fever), Moha (delusion), Tandrā (drowsiness), Avasāda (debility) and Prāṇasaṃśaya (leads to death without any doubt). Thousands of children will die from this disease and if life-saving treatment is done, some children will survive with debility continuing life-long with the appearance of severe bleeding disorders such as bloody diarrhoea, or bleeding through the hair pores and it is difficult to attain healthy state.

Sādhyāsādhyata of Masūrikā

⊙ त्वग्गता रक्तजाश्चैव पित्तजाः श्लेष्मजास्तथा । श्लेष्मपित्तकृताश्चैव सुखसाध्या मसूरिकाः ॥ (M.N.54/23)

Masūrikā that resides in Tvak (skin) and Raktaja, Pittaja and Śleṣmaja Masūrikā separately and Śleṣmapitta Masūrikā (together) are Sukha sādhya (easily curable).

⊙ वातजा वातपित्तोत्थाः श्लेष्मवातकृताश्च याः । कृच्छ्रसाध्यतमास्तस्माद्यत्नादेता उपाचरेत् ॥
असाध्याः सन्निपातोत्थास्तासां वक्ष्यामि लक्षणम् । प्रवालसदृशाः काश्चित् काश्चिज्जम्बूफलोपमाः ॥
लोहजालसमाः काश्चिदतसीफलसन्निभाः । आसां बहुविधा वर्णा जायन्ते दोषभेदतः ॥ (M.N.54/24-26)

Vātaja, Vātapittaja, Kaphavātaja Masūrikā are Kṛcchra sādhya (difficult to cure), so these should be treated with great effort. Sannipātaja Masūrikā is Asādhya (incurable) and their features are an eruption which is Pravāla sadṛśa (resembling the colour of coral), Jambūphalopāmāḥ (like the fruits of Jambu), Lohajālasamāḥ (like an iron sieve), Atasīphala sannibhā (like the fruits of Atasī) and of various colours depending on the predominance of doṣas.

⊙ कासो हिक्का प्रमेहश्च ज्वरस्तीव्रः सुदारुणः । प्रलापश्चारतिर्मूर्छा तृष्णा दाहोऽतिघूर्णता ॥
मुखेन प्रस्रवेद्रक्तं तथा घ्राणेन चक्षुषा । कण्ठे घुर्घुरकं कृत्वा श्वसित्यत्यर्थवेदनम् ॥
मसूरिकाभिभूतस्य यस्यैतानि भिषग्वरैः । लक्षणानि च दृश्यन्ते न दद्यादत्र भेषजम् ॥ (M.N.54/27-29)

The intelligent physician should not treat the patient of Masūrikā if the following features are manifested – Kāsa (cough), Hikkā (hiccup), Prameha (diabetes), Tīvrajvara (severe fever), Sudāruṇa pralāpa (severe delusion), Arati (restlessness), Mūrcchā (fainting), Tṛṣṇā (thirst), Dāha (burning sensation), Atidhūrṇatā (severe vertigo), Mukhena prasravedraktaṃ ghrāṇena cakṣuṣā (bleeding from the mouth, nose and eyes), Kaṇṭhe ghurghurakaṃ (murmuring sound in the throat), Śvāsa (difficulty in breathing) and Atyartha vedanā (severe pain).

⊙ मसूरिकाभिभूतो यो भृशं घ्राणेन निःश्वसेत् । स भृशं त्यजति प्राणांस्तृषार्तो वायुदूषितः ॥ (M.N.54/30)

In patients with Masūrikā, the presence of Bhṛśaṃ Ghrāṇena Niḥśvaset (very shallow breathing), Tṛṣārthā (severe thirst), and Vāyudūṣitaḥ (vitiated Vāta) may indicate a very soon death.

⊙ मसूरिकान्ते शोथः स्यात् कूपरे मणिबन्धके । तथांऽसफलके चापि दुश्चिकित्स्यः सुदारुणः ॥ (M.N.54/31)

In the final stage of Masūrikā, there is Kūrpara maṇibandhaka tathāaṃsaphalaka śotha (profound swelling in the elbow, wrist and shoulder joints). Moreover, this condition is incurable.

In Contemporary science,

Bṛhat Masūrikā, Laghu Masūrikā, and Romāntika can be respectively associated with Smallpox, Chickenpox, and Measles respectively. (Please refer Topic 19).

MCQ's

1. Doṣa predominance in Masūrikā
   - a) Vātapitta
   - b) Vātakapha
   - c) Pittakapha
   - d) Pittarakta
2. Śītalā is also known as
   - a) Masūrikā
   - b) Romāntikā
   - c) Carmapiḍaka
   - d) All of the above
3. According to Śārṅgadhara, Masūrikā is of .... types
   - a) 5
   - b) 7
   - c) 10
   - d) 14
4. Which type of Masūrikā is known as Carmapiḍaka
   - a) Vātaja
   - b) Pittaja
   - c) Kaphaja
   - d) Sannipātaja
5. Toyabudbuda saṅkaśā is the feature of
   - a) Tvakgata Masūrikā
   - b) Raktagata Masūrikā
   - c) Māṃsagata Masūrikā
   - d) Medogata Masūrikā
6. Maṇḍalākārā mṛdava unnatā piḍakā is the feature of
   - a) Tvakgata Masūrikā
   - b) Raktagata Masūrikā
   - c) Māṃsagata Masūrikā
   - d) Medogata Masūrikā
7. In Bṛhat Masūrikā, which day the Piḍaka appears on the Keśāntuṣu lalāṭe
   - a) 2nd day
   - b) 3rd day
   - c) 4th day
   - d) 5th day
8. In Bṛhat Masūrikā, when does the Piḍaka falls off from the skin
   - a) 7th day
   - b) 10th day
   - c) 12th day
   - d) 21st day

9. Doṣa predominance in Romāntika
   - a) Vātapitta
   - b) Vātakapha
   - c) Kaphapitta
   - d) Sannipātaja
10. Kaphapittaja Masūrikā is
    - a) Sukha sādhya
    - b) Kṛcchra sādhya
    - c) Yāpya
    - d) Asādhya

Answer Keys

1	d	2	a	3	d	4	d	5	a	6	d	7	b	8	c	9	c	10	a

◆

TOPIC 19

Fever

Introduction

Fever, also known as pyrexia, is defined as an elevation of body temperature exceeds the normal diurnal variation as a result of the change in the thermoregulatory center, located in the hypothalamus. The normal human body temperature is 37.0 °C (98.6 °F) and it varies from 0.5 to 1°C based on sex, age, time of day, activity levels, emotions, meal, sleep and during menstrual cycle in women physiologically. For an example, at early morning at 6.00 AM, temperature higher than 37.0°C (98.6°F) or at evening at 6.00 PM temperature higher than 37.6°C (99.6°F) is normally considered as fever. Pathological variation in the body temperature depends on the two conditions namely Hyperthermia and Hypothermia.

Hyperthermia is a medical emergency with high mortality and morbidity and it is defined as an abnormal increase in the body temperature due to failed thermo-regulation i.e. the body produces or absorbs more heat than it dissipates. The commonest cause of Hyperthermia includes Heat exhaustion, Heat stroke and adverse reaction to drugs and it is classified into Low grade fever (38- 39°C /100.4-102.2°F), moderate grade fever (39-40°C /102.2-104°F) and High grade fever (40- 42°C /104-107.6°F)

Hypothermia is also a medical emergency condition and it is defined as an unintentional drop in the body's core temperature below 35°C (95°F). Exposure to extreme cold, alcohol consumption, Drugs, Hypoglycemia, Hypothyroidism, Malnutrition, Injury, Sepsis, Shock and immobility are the main causes of Hypothermia and it is classified in to Mild (35- 33°C / 95-91.4°F), Moderate (33- 31°C / 91.4- 87.8°F) and Severe hypothermia (below 33°C / 87.8°F)

Hyperpyrexia is an extremely high body temperature where usually the rectal temperature is greater than or equal to 42.0°C (107.6°F) which ocurs as a result of severe infections, intracranial hemorrhage, sepsis, Kawasaki syndrome, neuroleptic malignant syndrome, drug overdose, serotonin syndrome and thyroid storm. If left untreated, it may damage various organs and results in death.

Causes of Fever:

Fever can arise from a diverse array of underlying causes. Infections are a leading cause, encompassing bacterial, viral, protozoal, fungal, and helminthic origins. These infections can range from common illnesses like pneumonia and urinary tract infections to more exotic diseases like malaria and toxoplasmosis.

Beyond infections, inflammatory conditions also trigger fever. Connective tissue diseases, such as rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE), are notable examples. Hypersensitivity reactions, including drug allergies, can also induce fever.

Trauma constitutes another significant cause, encompassing accidents, surgical interventions, and blunt injuries. Neurological disorders, including intracranial hemorrhage, can also precipitate fever. Endocrine disorders, such as thyroid storm and ovulation, represent additional triggers.

Lastly, neoplastic conditions, including various carcinomas, can cause fever, often as a symptom of the underlying malignancy or its treatment. This broad spectrum of potential causes underscores the importance of thorough diagnostic evaluation to identify the root cause of fever and provide effective management.

Patterns of Fever:

The pattern of the fever may be indicative of a particular diagnosis in many cases and therefore it is necessary to record the temperature periodically.

- **Continuous fever :** The temperature remains elevated throughout the day and the fluctuation of temperature does not exceed 0.6°C (1°F) e.g. lobar pneumonia, typhoid
- **Remittent fever :** The temperature remains elevated throughout the day and the fluctuation of temperature exceeds 0.6°C (1°F) e.g. infective endocarditis, brucellosis
- **Relapsing fever :** This is a recurring fever in which the episodes of fever lasts for several days, followed by an interval without fever, followed by another episode of fever e.g. Borrelia infections
- **Intermittent fever :** The elevated temperature touches the normal baseline in between the peaks or in other words, fever is present only for several hours during the day e.g. malaria, tuberculosis etc. Many intermittent fevers can occur with a periodicity.
    - **Quotidian fever :** Fever occurring every day due to Plasmodium falciparum.
    - **Tertian fever :** Fever occurring on alternate days due to Plasmodium ovale and vivax.
    - **Quartan fever :** Fever occurring once in every fourth day due to Plasmodium malariae.

- **Saddle back fever :** This is biphasic fever in which the fever lasts for 2-3days followed by a remission lasting for 2 days and the fever reappears and continues for 2-3 days e.g. Dengue fever
- **Pel – Epstein's fever :** This is a cyclic fever which is characterized by low grade fever for 3 -10 days followed by afebrile periods of the same duration e.g. Hodgkin's lymphoma

Pathophysiology of Fever

The pathophysiology of fever involves a complex immune response triggered by exogenous pyrogens, such as microbes, toxins, or tumors, that penetrate the body. This response activates host cells, including leukocytes and macrophages, to release endogenous pyrogens, comprising interleukin-1 (IL-1), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-alpha), and macrophage inflammatory protein-1 (MIP-1), among other cytokines.

These cytokines stimulate prostaglandin E2 (PGE2) production in the anterior hypothalamus, the body's thermoregulatory center. Increased PGE2 levels reset the body's temperature set point, initiating physiological responses to elevate body temperature. These responses include:

- Enhanced heat production through shivering and increased metabolism
- Reduced heat loss via vasoconstriction
- Behavioral responses, such as seeking warmth and rest

The resultant fever serves as a natural defense mechanism, creating an environment less conducive to microbial growth and replication.

Clinical evaluation of Fever

The clinical evaluation of fever necessitates a comprehensive approach aimed at identifying the underlying cause and guiding subsequent management strategies. This process begins with an extensive history-taking that emphasizes critical factors, including the duration, pattern, onset, associated symptoms, travel history, exposure to infectious diseases, medications, and pre-existing medical conditions.

A thorough physical examination is conducted to assess vital signs, general appearance, localized signs of infection, and organ-specific findings, all of which are crucial for pinpointing potential sources of infection. Laboratory investigations, including a complete blood count, blood cultures, urinalysis, chest radiography, serological tests, and molecular diagnostics, play a vital role in elucidating the underlying cause of the fever. The differential diagnosis encompasses a wide array of conditions, such as infectious diseases, inflammatory disorders, malignancies, endocrine disorders, and drug-induced fevers.

MEASLES

Introduction

Measles, also known as Rubeola, is a highly contagious and potentially life-threatening viral disease caused by the Paramyxovirus. This illness predominantly affects children under the age of ten, with a notably higher prevalence in those younger than five years. Pregnant women and individuals with compromised immune systems are also at increased risk. Globally, measles is estimated to impact approximately 20 million individuals annually. In the context of India, it accounted for 36% of the worldwide deaths attributed to this disease in 2020.

Incubation period: It is usually between 9 to 12 days

Mode of transmission: It spreads from person to person through droplet infection

Pathophysiology:

The pathophysiology of measles begins with the virus entering through the nasopharyngeal or conjunctival mucosa, where it binds to host cells using its H glycoprotein. Initial replication occurs in regional lymph nodes, infecting lymphocytes and leading to systemic spread. The virus targets lymphoreticular cells in vital organs like the spleen and liver, causing secondary viremia, which marks the start of the prodromal period, typically lasting six to seven days before the rash appears.

Infection triggers an inflammatory response with mononuclear reactions and giant cells containing inclusion bodies. Hemagglutinin binds to cellular receptors, while the fusion glycoprotein facilitates intercellular transmission, enabling viral replication and particle assembly. The virus also produces V and C proteins to inhibit interferon, aiding replication and immune suppression.

The immune response involves activation of CD4+ and CD8+ T cells, antibody production, and cytokine release. However, the immune suppression caused by the virus can compromise overall immune function, increasing susceptibility to secondary infections and complications.

Clinical features

The clinical features of measles are classified into 3 stages i.e. catarrhal stage, stage of rash and stage of convalescence.

- **In catarrhal stage,** the prodromal features of measles such as fever, malaise, cough, coryza, conjunctivitis, photophobia, swelling of eyelids, hoarseness of voice and presence of Pathognomonic Koplik's spots (small white spots surrounded by erythema appears on the buccal mucosa) are developed

- **In the stage of rash**, After 1 to 2 days of appearance of Koplik's spot, a brownish red macula-papular rash appears first at the back of the ears, forehead and spreads to face, neck, trunk and extremities with mild itching and musty smell. The other common features are generalized lymphadenopathy, toxemia, malaise, headache, sore throat, coated and furred tongue.
- **In the stage of convalescence**, the rash disappears in the same order with reduction in fever, malaise and toxemia.

Complications

The complications are more common in older children and adults, and it includes bronchitis, broncho or lobar pneumonia, otitis media, gastroenteritis, stomatitis, hepatitis, myocarditis, endocarditis, pericarditis and encephalomyelitis.

Diagnosis

Diagnosis is based on the clinical presentation and serological test including ELISA test which helps to detect the IgM and measles antibody and detection of virus in the blood, mucous, saliva, urine by the culture or Reverse transcription polymerase chain reaction (RT-PCR).

CHICKENPOX

Introduction

Chickenpox, also known as varicella, is a highly contagious viral infection caused by the varicella-zoster virus. It predominantly impacts children below the age of 10; however, adults may also be susceptible to the infection. Annually, approximately 140 million cases are reported worldwide, indicating its widespread nature. In India, from January 2015 to May 2021, there were 1,269 documented outbreaks of chickenpox, which resulted in 27,257 reported cases and led to 31 fatalities.

Incubation period: It is usually between 10 to 21 days

Mode of transmission: It is transmitted through droplets and direct contact with the infected person during the early stages of the eruption i.e. one or two days before the appearance of the skin lesion.

Pathophysiology:

The pathophysiology of chickenpox is initiated when the Varicella-zoster virus (VZV) enters the body via respiratory droplets or direct contact with lesions from infected individuals. Following entry, the virus replicates within respiratory epithelial cells and subsequently disseminates to regional lymph nodes. This process results in viremia, which allows the virus to spread to the skin and other organs.

Upon infecting skin cells, Varicella-zoster virus induces the formation of syncytia (multinucleated giant cells) and generates viral antigens that elicit an immune response. The immune system engages in this response through innate mechanisms, activating natural killer cells and macrophages, as well as adaptive mechanisms that stimulate CD4+ and CD8+ T cells along with the production of antibodies.

The viral damage to skin cells incites inflammation and the development of a rash that progresses through distinct stages: macules, papules, vesicles, pustules, and crusts. Neurological complications may arise if Varicella-zoster virus infects the dorsal root ganglia, resulting in symptoms such as headache and fever.

Histopathological analysis reveals intraepidermal vesicles and multinucleated giant cells within skin lesions, accompanied by inflammation and necrosis in the affected tissues. Additionally, cytokines, including interleukin-1, tumor necrosis factor-alpha, and interferon-gamma, contribute to inflammation and play a critical role in regulating viral replication.

Clinical features

The clinical features of chickenpox are classified into 2 stages i.e. stage of invasion and stage of eruption.

- In the **stage of invasion**, the prodromal symptoms like mild fever, headache, back pain, malaise and sore throat develops; followed by the appearance of enanthem over the palatal, buccal and pharyngeal mucosa. This stage usually lasts for 2 to 3 days.
- In the **stage of eruption**, the skin eruption first appears on the back and then it spreads in centripetal distribution i.e. most dense on face and trunk and sparse on limbs. The skin eruption appears in the form of small pink macules, and then progresses to papules, vesicles and pustules in 24 hours associated with severe itching which leads to rupture of the vesicles resulting in secondary bacterial infection which inturn causes cellulitis.

Complications

The most common complication of chickenpox is cerebral ataxia, pneumonitis, bronchitis, laryngitis, hepatitis, encephalitis, optic neuritis, keratitis and myocarditis.

Diagnosis

Diagnosis is based on the clinical presentation and DNA detection tests (PCR) as well as direct immunofluorescent antigen test which are used to confirm the diagnosis.

HERPES ZOSTER

Introduction

Herpes Zoster, commonly known as shingles, is a viral infection resulting from the reactivation of the varicella-zoster virus (VZV). Following a previous varicella infection, which is known as chickenpox, the virus remains dormant in the sensory ganglia of the cranial nerve or the dorsal root ganglia. While chickenpox predominantly affects children, herpes zoster primarily manifests in adults and the elderly population. Globally, the incidence of herpes zoster is approximately 3 to 5 cases per 1,000 person-years. This condition tends to become more prevalent with increasing age, with incidence rates peaking around 70 years.

Incubation period: The incubation period for Herpes Zoster typically ranges from 7-14 days, with an average of 10-13 days from reactivation of the varicella-zoster virus. Viral shedding occurs 1-5 days before the rash appears and lasts until the rash crusts over, usually 7-10 days.

Mode of transmission: Herpes Zoster is primarily spread through direct contact with lesions or rash of an infected person, airborne transmission through respiratory droplets containing the virus, and contaminated surfaces.

Pathophysiology:

The pathophysiology of Herpes Zoster involves the reactivation of the varicella-zoster virus (VZV), which remains latent in the dorsal root ganglia following an initial infection with chickenpox. Upon reactivation, the virus travels along the nerve pathways to the skin, resulting in a painful rash or blisters. Reactivation of the virus is frequently prompted by a decrease in varicella-zoster virus-specific cell-mediated immunity, which may arise due to ageing or conditions of immunosuppression. As systemic immunity diminishes, the virus re-emerges from its latent state and begins to replicate, leading to neuronal damage and subsequent inflammation.

The immune response plays a significant role in tissue damage and the development of the rash, with the release of cytokines intensifying the inflammatory response. The resultant rash is typically unilateral and follows a dermatomal distribution, often accompanied by symptoms such as pain, itching, and fever. Ageing, immunosuppression, psychological stress, and certain underlying medical conditions are contributing factors to the virus's reactivation.

Clinical features:

Herpes zoster typically manifests as a painful rash, or blisters localized to one side of the body, adhering to a specific dermatomal distribution. This rash progresses from macules to vesicles and ultimately forms crusts. Patients often experience accompanying symptoms, including fever, headache, fatigue, malaise, and regional lymphadenopathy. The rash generally develops within a single dermatome, encompassing the thoracic, cervical, trigeminal (including the ophthalmic branch), or lumbosacral regions.

The progression of the infection occurs in three distinct phases:

- **Pre-eruptive Stage:** This initial phase lasts a minimum of 48 hours before the appearance of any lesions. It is characterised by abnormal skin sensations or pain within the affected dermatome, accompanied by headaches, general malaise, and photophobia.
- **Acute Eruptive Phase:** Lasting 2 to 4 weeks, this phase is marked by the appearance of vesicles and an exacerbation of symptoms. Lesions progress from macules to painful vesicles, which may rupture, ulcerate, and subsequently crust over. Patients remain highly infectious until the lesions dry out, and they often experience severe pain that is frequently unresponsive to conventional analgesics.
- **Chronic Infection Phase:** Lasting more than 4 weeks, this phase is characterised by recurrent pain, paraesthesia, shock-like sensations, and dysesthesia. The pain may be debilitating and can persist for 12 months or more.

Additional symptoms may include burning, stabbing, aching pain, itching, tingling or numbness, mild fever, headache, fatigue, and malaise.

Complications

Herpes Zoster can lead to several complications, including postherpetic neuralgia (PHN), a persistent pain lasting more than four weeks after the rash resolves. Ocular complications, such as keratitis and uveitis, can also occur, potentially leading to vision loss. Additionally, neurological complications like encephalitis and meningitis may arise, and bacterial superinfections can develop.

Atypical presentations of Herpes Zoster include zoster sine herpete, characterised by pain without a rash, zoster multiplex, involving multiple dermatomes, and disseminated zoster, marked by a widespread rash.

Diagnosis

The diagnosis of Herpes Zoster typically begins with a clinical evaluation, including a medical history, physical examination, and characteristic rash distribution. A unilateral rash following a dermatomal pattern accompanied by pain, itching, or tingling is often indicative of Herpes Zoster.

Diagnostic tests confirm the presence of varicella-zoster virus (VZV). These include Tzanck smear, which detects multinucleated giant cells in vesicle fluid, viral culture to isolate varicella-zoster virus, polymerase chain reaction (PCR) to detect varicella-zoster virus DNA, and serology tests to identify varicella-zoster virus antibodies.

HAND, FOOT, AND MOUTH DISEASE

Introduction

Hand-foot-and-mouth disease (HFMD) represents a highly contagious viral infection attributed to various viruses within the Picornaviridae family, including Coxsackievirus A, Coxsackievirus B, and Enterovirus 71 (EV-71). This condition predominantly affects infants and children under the age of five; however, it can occasionally manifest in adults. Hand-foot-and-mouth disease is recognized as a global public health concern, particularly for young children, with an estimated incidence of 10 to 20 million cases annually. In India specifically, approximately 1.4 million children are reported to be affected each year.

Incubation Period: The incubation period of Hand-Foot-and-Mouth Disease (HFMD) typically ranges from 2-7 days, with an average of 3-5 days.

Mode of Transmission: It primarily spreads through direct contact, faecal-oral transmission, droplet transmission, and contaminated surfaces.

Pathophysiology:

The pathophysiology of Hand-Foot-and-Mouth Disease (HFMD) commences with viral entry through the gastrointestinal or upper respiratory tract, facilitated by infected hosts, vesicle fluid, or oral secretions. Subsequent replication in lymphoid tissues precedes primary viremia, where the virus enters the bloodstream and disseminates to various organs, including skin, mucous membranes, and the central nervous system.

As the virus invades epithelial cells, replication triggers cell lysis and necrosis, culminating in characteristic vesicle and ulcer formation. The host's immune response is activated, prompting inflammation and cytokine release, which mobilizes macrophages and T-cells. Factors influencing pathophysiology include virus strain, host immune status, age, susceptibility, and environmental factors like crowding and poor hygiene.

Clinical Features:

Hand-foot-and-mouth disease typically begins with mild symptoms, including low-grade fever, headache, fatigue, and loss of appetite. It typically presents with mouth or throat pain due to the enanthem (oral lesions). The characteristic vesicles are surrounded by a thin halo of erythema and eventually rupture, forming superficial ulcers with a grey-yellow base and erythematous rim.

The exanthem (skin rash) can manifest as macular (flat), papular (raised), or vesicular (blister-like) lesions, measuring 2-6 mm in size. Notably, these lesions are non-pruritic (non-itchy) and usually painless. They persist for approximately 10 days, often rupturing and leaving shallow, painless ulcers without scarring. The rash can involve various body parts, including the dorsum of hands and feet, buttocks, legs, and arms. Oral lesions commonly affect the buccal mucosa (inner cheeks), tongue, and soft palate.

Complications

Hand-foot-and-mouth disease can lead to various complications, including secondary bacterial infections, viral meningitis, encephalitis, respiratory distress, pneumonia, fever-induced seizures, dehydration, and skin infections.

In rare cases, HFMD may cause acute flaccid paralysis, Guillain-Barre Syndrome, myocarditis, pericarditis, aseptic meningitis, and encephalomyelitis. Long-term complications may arise, including neurological sequelae like cognitive impairment and behavioural changes and persistent neurological deficits such as weakness and numbness.

Diagnosis:

Diagnosis is primarily determined based on clinical presentation, characterised by fever, sore throat, difficulty swallowing, and vesicles or ulcers on the hands, feet, mouth, and buttocks. Laboratory tests may be employed to confirm the diagnosis, including viral isolation, serological assays, and molecular diagnostic techniques such as reverse transcription polymerase chain reaction (RT-PCR). The World Health Organization (WHO) proposes specific diagnostic criteria, including fever, a vesicular rash on the hands and feet, oral lesions, age under 10 years, and absence of other diagnoses.

RUBELLA

Introduction

Rubella, also known as German measles, is a highly contagious viral infection caused by a single-stranded, positive-sense, enveloped RNA virus. This infection predominantly affects children and non-immune young adults. According to the World Health Organization (WHO), rubella represents the leading vaccine-preventable cause of birth defects, with an estimated 100,000 infants worldwide being born with congenital rubella syndrome (CRS) each year. In 2022, approximately 17,865 cases of rubella were reported across 78 countries.

Incubation Period: The incubation period typically ranges from 12-23 days, averaging 14-21 days.

Mode of Transmission: Rubella is primarily transmitted through respiratory droplets, and contact with infected secretions like nasal discharge or saliva. It can also be transmitted from mother to fetus during pregnancy, leading to congenital rubella syndrome. Additionally, the virus may spread through contaminated surfaces and objects, known as fomites.

Pathophysiology:

The pathophysiology of rubella involves the replication of the rubella virus, a single-stranded RNA virus that belongs to the Togaviridae family, in the nasopharyngeal epithelium and regional lymph nodes after entry through respiratory droplets or contact with infected secretions. Viremia follows, spreading the virus to various organs, where it infects and damages cells, triggering an immune response characterized by pro-inflammatory cytokine release, activation of T-cells and macrophages, and production of neutralizing antibodies (IgM, IgG). In congenital rubella syndrome (CRS), maternal viremia during pregnancy allows the virus to cross the placenta, leading to fetal infection and congenital anomalies.

Unvaccinated individuals, pregnant women, immuno-compromised persons, healthcare workers, and close contacts of infected individuals are at high risk of transmission.

Clinical Features:

Rubella infection can be asymptomatic in 25-50% of cases. When symptoms occur, they typically include a prodromal illness characterized by low-grade fever, malaise, anorexia, headaches, sore throat, and adenopathy, followed by a rash consisting of pinpoint pink macules and papules that begin on the face and spread to the trunk and extremities, lasting approximately 3 days.

Congenital Rubella Syndrome (CRS) is associated with severe birth defects, including the classic triad of cataracts, congenital heart defects, and sensorineural deafness, as well as low birth weight, thrombocytopenic purpura, hemolytic anemia, hepatosplenomegaly, and meningoencephalitis.

Complications

Complications associated with rubella may include polyarthritis and polyarthralgia, which can affect up to 70% of adolescents and adult women. These conditions typically present symmetrically, primarily involving the wrists, fingers, knees, and ankles. In rarer instances, other manifestations may arise, such as thrombocytopenia, hemolytic anemia, myocarditis, pericarditis, hepatitis, orchitis, retinopathy, uveitis, Guillain-Barré syndrome, and post-infection encephalitis. Additionally, rubella infection during pregnancy poses significant risks, including miscarriage, intrauterine fetal demise, premature labor, intrauterine growth retardation, and congenital rubella syndrome.

Diagnosis:

The diagnosis of rubella is determined through clinical evaluation and serological testing. Clinically, rubella presents with a low-grade fever, rash, lymphadenopathy, and conjunctivitis.

Laboratory confirmation is achieved by detecting rubella-specific IgM antibodies in serum or saliva, indicating an acute infection; the presence of IgG antibodies suggests past infection or immunity. Virological analysis may involve isolating the virus from throat swabs, urine, or tissue samples, and molecular techniques such as reverse transcription-polymerase chain reaction (RT-PCR) are used to detect viral RNA.

Prenatal diagnosis of congenital rubella syndrome (CRS) employs ultrasound to identify fetal anomalies and amniocentesis or chorionic villus sampling (CVS) to test for viral RNA in amniotic fluid or placental tissue, respectively. Postnatally, CRS diagnosis relies on clinical findings and laboratory assessments to detect the rubella virus or antibodies in the newborn's blood or tissue.

MALARIA

Introduction

Malaria is a serious protozoal disease caused by Plasmodium parasites, transmitted primarily through the bites of infected female Anopheline mosquitoes. In the year 2022, the disease resulted in an estimated 249 million cases and approximately 608,000 fatalities on a global scale. India represents a notable contributor to the global malaria burden, accounting for approximately 1.7% of cases and 1.2% of deaths.

Incubation Period: The incubation period of malaria typically ranges from 7 to 30 days, depending on factors such as Plasmodium species, mosquito strain, human immunity, and parasite load. The incubation periods for various species are as follows:

- Plasmodium falciparum- 7-14 days, averaging of 10-12 days
- Plasmodium vivax - 10-17 days, averaging 12-14 days
- Plasmodium ovale - 12-18 days, averaging 14-16 days
- Plasmodium malariae - 12-18 days, averaging 14-16 days
- Plasmodium knowlesi, the most recently identified human malaria parasite, typically ranges from 6 to 14 days, with an average of 9-10 days.

Mode of Transmission: Malaria is primarily transmitted through the bites of infected female Anopheline mosquitoes. Additional transmission routes include contaminated blood transfusions, organ transplantation from infected donors, and congenital transmission from mother to child during pregnancy.

Pathophysiology:

The pathophysiology of malaria begins when an infected female Anopheles mosquito bites a human and injects Plasmodium sporozoites into the bloodstream. These sporozoites travel to the liver, where they invade liver cells (hepatocytes) and multiply asexually, forming mature schizonts. When these schizonts rupture, they release merozoites into the bloodstream, which then invade red blood cells (RBCs). Inside the RBCs, merozoites continue to multiply, leading to repeated cycles of RBC invasion, multiplication, and rupture.

As the host's immune system responds by producing antibodies and activating immune cells, the destruction of RBCs can trigger clinical symptoms such as fever, chills, anemia, and potential organ dysfunction. The severity of malaria can vary based on the Plasmodium species involved, the parasite load, and the host's immune status.

Clinical Features:

Malaria is characterized by a range of clinical features, often beginning with early symptoms such as fever, chills, rigors, headache, fatigue, nausea, vomiting, diarrhoea, muscle and joint pain, and abdominal pain. As the disease progresses, classic symptoms emerge, including periodic fever every 48-72 hours, paroxysms, anaemia, jaundice, and splenomegaly.

In severe cases, patients may experience cerebral malaria, severe anaemia, acute respiratory distress syndrome, renal failure, cardiovascular collapse, hemorrhagic complications, and metabolic acidosis.

The clinical presentation of malaria is influenced by the specific species of Plasmodium involved:

- **Plasmodium falciparum,** the most virulent species, is associated with high fever, chills, severe anemia, respiratory distress, and cerebral malaria, which may lead to multi-organ failure if not treated promptly.
- **Plasmodium vivax** infections tend to be milder, presenting with symptoms such as fever, chills, headache, and anemia; however, they may result in splenomegaly and periodic relapses due to dormant liver stages.
- **Plasmodium ovale** infections are similar to P. vivax, characterized by mild symptoms and the potential for relapses.
- **Plasmodium malariae** is known to induce a more chronic infection with mild manifestations, including fever, chills, and anemia, which may persist for several years without leading to severe illness.
- **Plasmodium knowlesi** found mainly in Southeast Asia, can result in severe and rapidly progressing malaria, exhibiting high fever, chills, severe anemia, and elevated parasitemia levels, culminating in complications akin to those caused by P. falciparum.

Complications

Complications of malaria can be severe and life-threatening. Among these, cerebral malaria is particularly concerning, as it can lead to seizures, coma, and irreversible brain damage. Hematological complications can manifest as severe anemia, hemorrhage, and disseminated intravascular coagulation (DIC).

Respiratory complications, such as acute respiratory distress syndrome (ARDS) and pulmonary edema, may result in respiratory failure. Renal complications—including acute kidney injury (AKI), nephrotic syndrome, and hematuria—pose the risk of permanent damage to kidney function. Furthermore, cardiovascular complications may occur, resulting in hypotension, cardiac arrest, and arrhythmias.

Metabolic complications, including hypoglycemia, metabolic acidosis, and electrolyte imbalances, are also possible. Other significant concerns encompass sepsis, organ failure affecting the liver, spleen, and pancreas, as well as dehydration and secondary infections caused by bacterial, viral, or fungal agents.

Pregnant women are at increased risk of complications, including miscarriage, stillbirth, and low birth weight. Consequently, prompt diagnosis and treatment are essential to mitigating these complications and reducing mortality rates. If malaria remains untreated, it can lead to long-term health consequences, such as cognitive impairment, neurological damage, and increased susceptibility to future infections.

Diagnosis

Diagnosis is based on clinical evaluation, laboratory tests, and imaging studies. Clinically, symptoms such as fever, chills, rigors, headache, and flu-like illness are significant indicators, particularly in individuals with a history of travel to endemic regions.

Laboratory tests play a critical role in confirming the diagnosis. Peripheral blood smears are utilized to detect parasites through microscopy. Rapid diagnostic tests (RDTs) facilitate the identification of specific antigens or antibodies, while polymerase chain reaction (PCR) is instrumental in species identification. Additionally, serological tests are employed to detect the presence of antibodies. Imaging studies, including ultrasound and CT scans, are conducted to evaluate potential complications such as splenomegaly or cerebral oedema.

FILARIASIS

Introduction

Filariasis, commonly known as lymphatic filariasis or elephantiasis, is a parasitic disease caused by infection with any of three species Wuchereria bancrofti, Brugia malayi, and Brugia timori. Globally, lymphatic filariasis affects over 657 million people in 39 countries, with an estimated 120 million people infected. In India, approximately 50-60 million people are infected, with 65% of the population living in endemic areas, primarily in southern, eastern, and northeastern states.

Incubation Period: The incubation period of Filariasis typically ranges from 8 to 16 months, but in some cases, although it can be as short as 4 weeks in some cases. The incubation periods for the various species are as follows:

- Wuchereria bancrofti: 8-16 months (range: 6-18 months)
- Brugia malayi: 3-6 months (range: 2-12 months)
- Brugia timori: 3-6 months (range: 2-12 months)

Mode of Transmission: It is primarily transmitted through the bite of an infected mosquito, primarily Culex quinquefasciatus, Anopheles, Aedes, and Mansonia.

Pathophysiology:

The pathophysiology begins with a mosquito bite that transmits infective larvae (L3) into human skin. These larvae subsequently penetrate the dermal layer and enter lymphatic vessels, migrating toward lymph nodes. Within these nodes, the larvae mature into adult worms, referred to as macrofilariae, which then produce microfilariae (mf) that circulate in the bloodstream. The host's immune response incites chronic inflammation within the lymph nodes and vessels, resulting in lymphatic obstruction and scarring, which ultimately leads to lymphoedema, characterized by localized swelling.

The adult worms obstruct lymphatic flow mechanically, causing damage to the vessels, valves, and lymph nodes. This obstruction may give rise to various complications, including elephantiasis, which manifests as severely enlarged limbs, Hydrocele involving scrotal swelling, chyluria that results in urine discoloration, lymphangitis indicating inflammation of lymph vessels, and skin thickening along with fibrosis. Cell-mediated immunity (CMI) is instrumental in regulating the infection, while antibody production plays a vital role in the clearance of microfilariae. Pathological alterations associated with the disease include the enlargement and fibrosis of lymph nodes, dilation and obstruction of lymphatic vessels, and thickening of the skin and subcutaneous tissue.

Clinical features:

Lymphatic filariasis presents a range of clinical features, progressing from acute to chronic stages. Initially, patients may experience symptoms such as fever, chills, headache, muscle and joint pain, swollen lymph nodes, and skin rashes or lesions. As the disease progresses to its chronic stage, symptoms may include lymphoedema (swelling of the limbs, genitals, or breasts), elephantiasis (severe enlargement of the limbs), hydrocele (swelling of the scrotum), chyluria (discoloration of urine), and lymphangitis (inflammation of lymphatic vessels).

Furthermore, complications may arise, including skin thickening and fibrosis, recurrent infections (such as cellulitis and abscesses), and severe health issues like renal failure, cardiovascular disease, respiratory problems, and mental health disorders (including depression and anxiety). In advanced cases, individuals may endure significant disability and face social stigma.

The clinical manifestations of lymphatic filariasis can be categorized into four distinct groups: asymptomatic, microfilaraemic (indicating the presence of microfilariae in the blood), clinical filariasis (characterized by lymphoedema and hydrocele), and **chronic filarial disease** (such as elephantiasis). The stages of lymphoedema range from latent (absence of swelling) to severe (extensive swelling and fibrosis), encompassing mild (reversible swelling) and moderate (irreversible swelling) stages.

Diagnosis

Diagnosis is based on clinical evaluation, laboratory tests, and imaging studies. Clinically, diagnosis is based on symptoms such as lymphoedema, hydrocele, and elephantiasis.

Laboratory tests include microscopy to detect microfilariae in blood or lymphatic fluid, antigen detection tests to identify filarial antigens in blood or urine, and PCR for detecting parasite DNA in blood or tissue.

Imaging studies such as ultrasound, CT, or MRI scans help visualize lymphatic damage. Additional diagnostic tools include lymphoscintigraphy to trace lymphatic flow, Doppler ultrasound to assess lymphatic vessel function, and fine-needle aspiration biopsy to examine lymph nodes or tissue samples.

The WHO recommends a two-step approach: screening with rapid diagnostic tests (RDTs) or antigen detection tests, followed by confirmation with microscopy, PCR, or imaging studies.

INFLUENZA

Introduction

Influenza, also known as flu, is an acute viral infection of the respiratory tract caused by influenza virus A and B. Annually, approximately one billion cases of seasonal influenza are documented, resulting in 3 to 5 million severe illnesses and between 290,000 and 650,000 respiratory fatalities. Individuals at increased risk include pregnant women, children under the age of five, older adults, and persons with chronic medical conditions.

Incubation period : It ranges from 1 to 4 days with an average of about 48 hours.

Mode of Transmission : It is transmitted through droplet infection.

Pathophysiology:

The pathophysiology of influenza involves a multi-stage process. Influenza viruses enter the body through the respiratory tract, typically via inhalation of droplets from an infected person's cough or sneeze, attaching to epithelial cells using hemagglutinin (HA) proteins. Once inside, the virus releases its RNA, replicating and transcribing viral proteins using the host cell's machinery. New viral particles assemble and bud off, spreading to adjacent cells. The infection triggers an immune response, releasing cytokines and interferons, leading to inflammation and characteristic symptoms like fever, muscle pain, and fatigue. The virus-induced destruction of epithelial cells and immune response-driven inflammation damage the respiratory tract's mucosal lining, increasing the risk of secondary bacterial infections, such as pneumonia. The widespread immune response and inflammatory mediators release also cause systemic symptoms like fever, chills, and muscle aches, alongside respiratory symptoms like cough and sore throat. This complex interplay between viral replication and host immune response underlies the clinical manifestations of influenza.

Clinical features:

The clinical features are sudden onset of high fever, chills, coryza, sneeze, sore throat, malaise, shortness of breath, dry and persistent cough, headache, mild conjunctivitis, photophobia, pain in the eyes, nausea, vomiting and abdominal pain.

Complications:

Bacterial Pneumonia is the most common complication of influenza; rare extra-pulmonary complications include myositis, myocarditis, pericarditis, encephalitis and reye syndrome.

Diagnosis:

Diagnosis is based on clinical evaluation, rapid influenza test, naso- pharyngeal swab culture and Reverse transcriptase–polymerase chain reaction (RT-PCR); pulse oximetry helps to rule out hypoxemia and imaging studies such as chest x-ray, CT and MRI helps to diagnose the pneumonia.

DENGUE FEVER

Introduction

Dengue is an acute, infectious arboviral disease caused by the dengue virus, belonging to the genus Flavivirus, and transmitted by the bite of infected female Aedes mosquito specifically Aedes aegypti, Aedes polynesiensis, Aedes scutellaris, and Aedes albopictus. The dengue virus comprises four distinct serotypes: DEN-1, DEN-2, DEN-3, and DEN-4. Although these serotypes elicit similar clinical manifestations, DEN-2 is particularly linked to severe forms of the disease, including Dengue Hemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS). The colloquial term "Break-bone Fever" aptly describes dengue due to the intense muscular and joint pain it induces.

Globally, it is estimated that dengue infections range between 100 million to 400 million annually, resulting in approximately 22,000 fatalities. In India, dengue remains endemic in various regions and frequently results in annual epidemics. The nation experiences two peaks of dengue activity each year: one occurring from January to March and another during the post-monsoon season. In 2022, India documented over 63,000 reported cases of dengue.

Incubation period : The incubation period is 4 to 7 days but it ranges from 3 to 15 days

Pathophysiology:

The pathophysiology of dengue involves a complex interplay between viral replication, host immune response, and vascular endothelium damage. Dengue virus enters host cells through receptor-mediated endocytosis, replicating viral RNA and producing viral proteins. The immune response is characterized by innate immunity activation, antibody-dependent enhancement, and cellular immunity. However, an excessive cytokine storm leads to vascular endothelium damage, increased permeability, and plasma leakage. This results in thrombocytopenia, coagulopathy, and hemorrhagic manifestations. Disease severity factors include virus serotype (DEN-2), host genetics, immune response, age, sex, and pre-existing conditions. The pathological changes include hemorrhagic manifestations, hepatic and splenic enlargement, and vascular endothelial damage.

Clinical features

The clinical features of classical dengue fever are sudden onset of high fever, chills, headache, myalgia, arthralgia, erythematous skin rash, retro-orbital pain on eye movement, anorexia, nausea, vomiting and loss of taste. The temperature is typically rises up to 39 to 40°C followed by rapid defervescence with profuse sweating for few hours to 2 days followed by second phase of fever (saddle back pattern) for 1 to 2 days. On examination, relative bradycardia and lymphadenopathy may be seen.

Dengue hemorrhagic fever (DHF) is characterized by sudden onset of fever, headache, myalgia, arthralgia, anorexia, nausea and vomiting, just like of classical dengue fever. But the temperature falls below normal and produces severe abdominal pain and tenderness, oedema, lethargy, restlessness and hemorrhagic tendencies develops as a petechiae, ecchymoses, purpura, haematemesis, gum bleeding, and malena. On examination, hepatomegaly, pleural effusion or ascites may be seen. WHO classified Dengue hemorrhagic fever into four grades I-IV. Grade I and II reveals the easy bruising and presence of spontaneous bleeding into the skin respectively. Grade III and IV shows the clinical evidence of shock and shock with low blood pressure and pulse respectively.

Dengue shock syndrome (DSS) is characterized by features of dengue hemorrhagic fever (Grade III and IV) with cold clammy extremities and associated with circulatory failure including hypotension, weak pulse, cyanosis, tachypnoea and hepatomegaly.

Diagnosis:

Diagnosis is based on the blood investigations and serological test. Blood investigations reveals Thrombocytopenia <100,000/mm^3, leucopenia, neutropenia and deranged prothrombin time and partial thromboplastin time. Serological test includes Reverse transcriptase–polymerase chain reaction (RT-PCR) and ELISA test for IgG and IgM Test.

LEPTOSPIROSIS

Introduction

Leptospirosis is a zoonotic infection caused by various serotypes of the spirochete bacteria within the genus Leptospira. This disease is transmitted from animals to humans, either directly or indirectly. Human infection primarily results from exposure to Leptospira icterohaemorrhagiae (associated with rodents), Leptospira canicola (linked to domestic dogs and pigs), and Leptospira hardjo (found in cattle), with rodents serving as the predominant reservoirs.

Infection occurs through direct contact with the urine or tissues of infected animals or indirectly via contaminated water, soil, or vegetation. The bacteria can penetrate the human body through breaches in the skin, particularly on the feet and extremities, as well as through exposed mucosal surfaces in the eyes, nose, or mouth.

Leptospirosis is typically prevalent in temperate climates during late summer or early autumn in Western nations, while in tropical regions, it tends to occur during the rainy season. The World Health Organization estimates that each year there are approximately 873,000 cases of leptospirosis, resulting in over 40,000 fatalities.

Incubation Period : The incubation period is between 2 to 26 days but most often it ranges from 7 to 13 days.

Pathophysiology:

The pathophysiology of leptospirosis involves the infiltration of Leptospira bacteria into the host organism through exposure of the skin or mucous membranes to contaminated water, soil, or animal urine. Upon entry, the bacteria breach blood vessel walls, resulting in endothelial damage and the activation of inflammatory responses. The lipopolysaccharide (LPS) component of Leptospira instigates a cytokine storm, which increases vascular permeability and can lead to haemorrhage and organ dysfunction.

The bacteria then spread to various organs, including the kidneys, liver, lungs, and central nervous system, causing renal failure due to tubular necrosis and interstitial nephritis, hepatic dysfunction indicated by elevated liver enzymes, pulmonary hemorrhage and acute respiratory distress syndrome (ARDS), and meningitis and encephalitis. The immune response, involving both humoral and cell-mediated immunity, plays a crucial role in eradicating the bacteria.

Clinical features:

Leptospirosis occurs in two phase namely leptospiraemic phase and immune phase.

- **Leptospiraemic phase** is characterized by abrupt onset of fever, chills, headache, severe myalgia, nausea, vomiting and conjunctival suffusion. Sometimes macular or maculo-papular skin rashes appear on the trunk. Lymphadenopathy, hepatomegaly and splenomegaly are uncommon. This phase lasts for 3 to 9 days followed by defervescence with asymptomatic period of 1 to 3 days.

- Then the second phase (**immune phase**) of the disease develops with fever, headache, neck rigidity, photophobia and appearance of antibodies in the serum. In this phase, the complications like myelitis, encephalitis, optic neuritis, uveitis, iridocyclitis and peripheral neuropathy develops.

Weil's syndrome (icteric leptospirosis) is a severe form of Leptospirosis characterized by continuous fever, jaundice, renal failure associated with hemorrhagic manifestations.

Diagnosis:

Diagnosis is based on the blood investigation, blood culture and Serological test. Blood test shows increased WBC and ESR; decreased platelet and haemoglobin level; increased SGOT, SGPT and alkaline phosphatase; blood, cerebrospinal fluid and urine culture is done to isolate the organism; serological test reveals positive microscopic agglutination test and ELISA IgM and slide agglutination test.

CHIKUNGUNYA

Introduction

Chikungunya is an acute infectious disease caused by Chikungunya virus (CHIKV), a member of the genus Alpha virus within the family Togaviridae. This disease is primarily transmitted through the bites of infected female Aedes mosquitoes, specifically Aedes aegypti and Aedes albopictus. Initially identified in Tanzania in 1952, CHIKV has subsequently disseminated on a global scale, with outbreaks documented in Asia, Europe, and the Americas, particularly in tropical and subtropical regions. As of 2024, there have been approximately 460,000 reported cases and 170 fatalities worldwide.

Incubation period: The incubation period is between 2 to 12 days

Pathophysiology:

The pathophysiology of chikungunya begins with the entry of the Chikungunya virus (CHIKV) into the human body through the bite of an infected Aedes mosquito. Upon entry, the virus selectively targets fibroblasts, muscle cells, and macrophages for replication. This process elicits a formidable immune response characterized by the release of pro-inflammatory cytokines, which results in symptoms such as fever and significant joint pain. Subsequently, the virus disseminates through the bloodstream, leading to viremia and potential persistenc in joint tissues, which may culminate in chronic arthralgia and arthritis in certain individuals. The inflammatory response and immune activation are central to the acute manifestations of chikungunya, including elevated fever, myalgia, headache, and rash. Sustained inflammation may contribute to enduring joint pain and discomfort.

Clinical features:

The clinical features of chikungunya typically commence with an abrupt onset of fever, frequently accompanied by chills, rigors, and a maculopapular rash. Severe pain and stiffness in the joints, particularly in the knees, ankles, shoulders, wrists, and interphalangeal joints, are distinguishing characteristics of the condition. Myalgia and headaches are also prevalent. These symptoms generally persist for a duration of weeks to months but may evolve into chronic conditions, particularly in individuals who are positive for HLA-B27. In some instances, conjunctivitis and neurological manifestations may arise. The chronic phase is marked by persistent joint pain and stiffness, which can lead to chronic arthralgia, arthritis, and osteoarthritis. Moreover, complications may include chronic joint damage, neuropathic pain, and psychological distress.

Diagnosis:

Diagnosis is based on the Laboratory investigation and Imaging studies. Laboratory diagnosis comprises serological tests, including enzyme-linked immunosorbent assay (ELISA), and Immunofluorescence assay (IFA), aimed at detecting IgM and IgG antibodies, in addition to molecular diagnostics utilizing RT-PCR for the identification of viral RNA. Viral isolation from blood or tissue samples may also be performed as part of the diagnostic process. The presence of IgM antibodies signifies a recent infection, while the detection of IgG antibodies indicates a prior infection or established immunity. A positive result from RT-PCR confirms ongoing viral replication.

Imaging studies, such as radiographs, ultrasound, or MRI, may be employed to evaluate joint damage and inflammation, identifying conditions such as bone erosions, joint space narrowing, and the formation of osteophytes.

TYPHOID FEVER

Introduction

Typhoid fever also known as enteric fever, is a potentially fatal multisystemic illness primarily caused by the Gram-negative bacterium Salmonella enterica serotype Typhi. Related serovars, including Paratyphi A, B, and C, contribute to the incidence of the disease to a lesser extent. Annually, it is estimated that 21 million individuals worldwide contract typhoid fever, resulting in approximately 161,000 fatalities. In India, the incidence rate is reported at 360 cases per 100,000 person-years, culminating in approximately 4.5 million cases and 8,930 deaths each year.

The prevalence of typhoid fever is significant across all age groups, with the highest incidence observed among children aged 5 to 13 years in endemic regions. Epidemiological studies indicate that males are more frequently affected; conversely, females are more likely to become chronic carriers of the infection.

Incubation Period – usually from 10-14 days but varies from 3 to 21 days.

Mode of Transmission – Faecal-oral route by food and water contaminated with faeces or urine of the infected person.

Pathophysiology:

The pathophysiology of typhoid fever initiates with the ingestion of food or water contaminated with Salmonella Typhi. Upon entering the gastrointestinal tract, this bacterium invades the intestinal mucosa and is capable of surviving within intestinal macrophages. Subsequently, it enters the bloodstream through intestinal lymphatics, disseminating to various organs, including the liver, spleen, and lymph nodes, which results in inflammation and tissue damage.

In response to the infection, the immune system is activated, engaging both innate and adaptive immunity. However, the formation of immune complexes may exacerbate tissue damage. Organ-specific effects are observed, such as ulceration and perforation of the intestines, hepatitis, splenomegaly, and, in rare instances, meningitis or encephalitis.

Salmonella Typhi generates both endotoxins and exotoxins, stimulating the release of cytokines, including TNF-α, IL-1β, and IL-6. This cascade leads to fever and inflammation. Potential complications may include intestinal perforation, hemorrhage, sepsis, and osteomyelitis. Furthermore, in certain cases, Salmonella Typhi can establish a chronic presence in the gallbladder, resulting in long-term carriage and shedding of the bacteria in feces, thereby contaminating the environment.

Clinical features:

The clinical features of typhoid fever typically unfold over four weeks. Initially, symptoms appear in the first week, characterized by a continuous, prolonged fever in a step-ladder pattern, accompanied by malaise, headache, anorexia, cough, epistaxis, sore throat, relative bradycardia, abdominal pain, and constipation or pea soup diarrhoea. Around the seventh day, a distinctive rose spot rash (pink papules) emerges on the chest and abdomen, lasting 3-4 days.

In the second week, the temperature suddenly drops, followed by an increased pulse, decreased blood pressure, mild hepatosplenomegaly, and the onset of the "Typhoid state" - a condition marked by prolonged apathy, toxaemia, delirium, or coma.

The third week is critical, with potential complications arising, such as intestinal bleeding, intestinal perforation, psychosis, meningismus, hepatitis, bronchitis, pneumonia, myocarditis, cholecystitis, nephritis, osteomyelitis, coma, and death.

By the end of the third week, the fever begins to subside, marking the start of convalescence, which extends into the fourth week. During this period, patients gradually recover, although some may experience lingering fatigue and weakness.

Diagnosis:

Diagnosis is based on the clinical features, physical examination, and laboratory assessments. Blood tests typically indicate normocytic normochromic anaemia, leucopenia, and a positive blood culture within the initial week of infection. In subsequent weeks, stool and urine cultures are generally positive by the second and third weeks, respectively, with bone marrow cultures occasionally yielding positive results as well.

The Widal test helps to detect the levels of agglutinating antibodies against the O and H antigens. The IDL Tubex test identifies IgM O9 antibodies, while the Typhidot test confirms the presence of specific IgM and IgG antibodies. Additionally, the Typhidot-M test is employed specifically to detect IgM antibodies.

COMMON REGIONAL DISORDERS PRESENTING WITH FEVER

Introduction

Fever is a prevalent symptom and a key indicator of various underlying medical conditions, including infectious and non-infectious disorders. Regional disorders exhibit distinct epidemiological patterns, affect specific geographical regions, and are influenced by different climatic, environmental, and socioeconomic factors. This chapter will discuss the common diseases presenting with fever in various geographical areas, excluding previously discussed conditions.

Tropical Regions

- **Zika Virus:** Found primarily in Central and South America, the Caribbean, and Southeast Asia, the Zika virus is transmitted by Aedes mosquitoes. Symptoms often include mild fever, rash, conjunctivitis, and joint pain. In severe cases, it can lead to neurological disorders and congenital disabilities.
- **Yellow fever:** Caused by the Yellow Fever virus, transmitted by Aedes mosquitoes. It is prevalent in sub-Saharan Africa and South America. Symptoms include high fever, chills, headache, muscle pain, and jaundice. Severe cases can lead to haemorrhaging, organ failure, and death.

Southeast Asia

- **Japanese Encephalitis:** This disease is caused by the Japanese Encephalitis virus and transmitted by Culex mosquitoes. It is common in rural areas of Asia. Symptoms include fever, headache, seizures, and neurological damage. Severe cases can lead to coma and death.
- **Scrub Typhus:** This disease is caused by Orientia tsutsugamushi and is transmitted by chigger mites. Symptoms include fever, headache, rash, and swelling at the bite site.

Desert region

- **Brucellosis:** Caused by Brucella species, this disease is transmitted through unpasteurized dairy products or contact with infected animals. It is common in the Middle East and North Africa, and symptoms include fever, fatigue, joint pain, and swelling.
- **Sandfly Fever:** Caused by Phlebovirus and spread by sandflies, symptoms include fever, headache, joint pain, and swelling.

Indian Subcontinent:

- **Kyasanur Forest Disease:** This disease is caused by the KFD virus and is transmitted through tick bites. It is endemic in some areas of India, particularly in the Karnataka, Maharashtra, and Kerala forests. Symptoms include fever, headache, joint pain, and haemorrhaging.
- **Kala-azar (Visceral leishmaniasis):** Caused by Leishmania donovani and transmitted by infected sandflies. Kala-azar is most prevalent in India, Bangladesh, Nepal, Sudan, Brazil, and other tropical and subtropical regions. Symptoms include fever, weight loss, lymphadenopathy, anaemia and hepatosplenomegaly.
- **Nipah Virus:** This virus is transmitted through contact with infected bats or contaminated food. It is common in India and Southeast Asia, and symptoms include fever, headache, respiratory distress, and neurological damage.

Latin America:

- **Chagas Disease:** Caused by Trypanosoma cruzi and transmitted by triatomine bugs. Common in Central and South America, symptoms include fever, swollen lymph nodes, cardiac issues, and gastrointestinal problems.
- **Venezuelan Hemorrhagic Fever:** Caused by the Guanarito virus, transmitted through contact with infected rodents. It is common in Venezuela, and symptoms include fever, bleeding, organ failure, and death.

Sub-Saharan Africa

- **Lassa fever:** Caused by the Lassa virus, transmitted through contact with rodent excreta. It is commonly seen in West Africa, and symptoms include fever, headache, joint pain, and haemorrhaging.
- **Ebola Virus Disease:** Caused by the Ebola virus and transmitted through contact with the bodily fluids of infected individuals. It is common in Central and West Africa, and symptoms include fever, vomiting, diarrhoea, and bleeding.
- **Rift Valley Fever:** Caused by the Rift Valley Fever virus and transmitted by mosquitoes. It is common in East and Southern Africa, and symptoms include fever, headache, joint pain, and haemorrhaging.

MCQ's

1. What is the causative agent of measles

a) Paramyxovirus b) Plasmodium

c) Leptospira d) Trypanosoma

2. What is the usual incubation period for measles

a) 3-5 days b) 5-7 days

c) 9-12 days d) 14-21 days

3. Which clinical feature is characteristic of the catarrhal stage of measles

a) High fever b) Koplik's spots

c) Rash on the face d) Severe joint pain

4. What is the causative agent of chickenpox?

a) Herpes Simplex Virus b) Epstein-Barr Virus

c) Varicella-Zoster Virus d) Cytomegalovirus

5. What is the usual incubation period for chickenpox?

a) 3-5 days b) 7-9 days

c) 10-21 days d) 14-28 days

6. Which is a common complication of chickenpox?

a) Myocarditis b) Pneumonitis

c) Pancreatitis d) Meningitis

7. How is Herpes Zoster transmitted

a) Through contaminated food

b) Through mosquito bites

c) Through direct contact with lesions or rash

d) Through direct contact with infected blood

8. Where does the virus remain dormant before reactivation

a) Lymph nodes

b) Bone marrow

c) Sensory ganglia of cranial nerve or dorsal root ganglia

d) Liver

9. What is a common complication of Herpes Zoster?

a) Postherpetic neuralgia (PHN)

b) Bronchitis

c) Pancreatitis

d) Meningitis

10. What viruses are responsible for Hand-foot-and-mouth disease (HFMD)

a) Herpes Simplex Virus

b) Coxsackievirus A, Coxsackievirus B, and Enterovirus 71 (EV-71)

c) Epstein-Barr Virus

d) Cytomegalovirus

11. Which symptoms are characteristic of the clinical features of HFMD?

a) Severe joint pain and swelling

b) Painful skin rash and high fever

c) Low-grade fever, headache, fatigue, and vesicular rash

d) Chronic cough and wheezing

12. What is the causative agent of rubella?

a) Epstein-Barr Virus b) Cytomegalovirus

c) Varicella-Zoster Virus d) Single-stranded RNA virus

13. What is a significant risk of rubella infection during pregnancy?
a) Chronic liver disease
b) Postherpetic neuralgia
c) Congenital rubella syndrome (CRS)
d) Chronic kidney disease

14. Which symptom is characteristic of rubella?
a) Severe joint pain and swelling
b) Painful skin rash and high fever
c) Pinpoint pink macules and papules rash
d) Chronic cough and wheezing

15. Which species of Plasmodium is the most virulent and associated with severe malaria?
a) Plasmodium vivax
b) Plasmodium malariae
c) Plasmodium falciparum
d) Plasmodium ovale

16. What is the usual incubation period for Plasmodium falciparum?
a) 3-5 days
b) 7-14 days
c) 10-21 days
d) 14-28 days

17. Which Plasmodium species can cause periodic relapses due to dormant liver stages?
a) Plasmodium falciparum
b) Plasmodium vivax
c) Plasmodium malariae
d) Plasmodium knowlesi

18. What is a significant complication of cerebral malaria?
a) Chronic liver disease
b) Severe anaemia
c) Hypotension
d) Seizures and coma

19. Which species of mosquito primarily transmits filariasis?
a) Aedes aegypti
b) Culex quinquefasciatus, Anopheles, Aedes, and Mansonia
c) Tsetse fly
d) Sandfly

20. Which clinical feature is characteristic of the chronic stage of lymphatic filariasis?
a) High fever and chills
b) Skin rashes or lesions
c) Elephantiasis (severe enlargement of the limbs)
d) Severe joint pain and swelling

21. Which serotype of the dengue virus is particularly linked to severe forms like Dengue Hemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS)?
a) DEN-1
b) DEN-2
c) DEN-3
d) DEN-4

22. Which mosquito species primarily transmits dengue?
a) Anopheles mosquitoes
b) Culex mosquitoes
c) Aedes mosquitoes
d) Tsetse fly

23. What is a significant complication of Dengue Hemorrhagic Fever (DHF)?
a) Chronic liver disease
b) Severe abdominal pain and hemorrhagic tendencies
c) Polyarthritis
d) Hypotension

24. Which animals are primarily associated with transmitting Leptospira icterohaemorrhagiae to humans?
a) Domestic dogs and pigs
b) Cattle
c) Rodents
d) Birds

25. What are the significant complications of Weil's syndrome?
a) Skin rash and itching
b) Continuous fever, jaundice, and renal failure
c) Severe joint pain and swelling
d) Chronic cough and wheezing

26. What is the usual incubation period for chikungunya?
a) 1-3 days
b) 2-12 days
c) 14-21 days
d) 21-30 days

27. What are common clinical features of chikungunya?
a) Severe joint pain, fever, myalgia, and rash
b) Chronic cough and wheezing
c) Persistent rash and itching
d) High fever, chills, and severe headache

28. What are common clinical features of typhoid fever in the first week?
   a) Severe joint pain and swelling
   b) Persistent rash and itching
   c) Continuous, prolonged fever, headache, anorexia, and abdominal pain
   d) Chronic cough and wheezing

29. What distinctive feature appears around the seventh day of typhoid fever?
   a) Severe joint pain
   b) Rose spot rash on the chest and abdomen
   c) Persistent itching
   d) Chronic cough

Answer Keys

1	a	2	c	3	b	4	c	5	c	6	b	7	c	8	c	9	a	10	b
11	c	12	d	13	c	14	c	15	c	16	b	17	b	18	d	19	b	20	c
21	b	22	c	23	b	24	c	25	b	26	b	27	a	28	c	29	b		

Short Answer Questions

1. Describe Measles
2. Describe Herpes zoster
3. Explain Hand foot mouth disease
4. Explain Chickenpox
5. Explain Malaria
6. Explain Dengue fever
7. Explain Leptospirosis
8. Explain Filariasis

◆

TOPIC 20

Pāṇḍu

Introduction

In Āyurveda, nomenclature of the diseases has its own importance; The disease has been named on the basis of type of Ruja (pain), Varṇa (color), Samutthāna (etiology), Sthāna (site), Saṃsthāna (symptoms) or Nāma (name). Pāṇḍu roga has been described on the basis of its Varṇa (colour). Even Ācārya Cakrapāṇi explained Pāṇḍu as **Pradhānena Varṇenopalakṣito rogaḥ** (the disease that is prominently characterized by its colour).

Nirūkti and Paribhāṣā

- सितपीतसमायुक्तः पाण्डुवर्णः प्रकीर्त्तितः ॥ इति सुभूतिः ॥ (SKD)

Pāṇḍu varṇa is an equal mixture of Sita (white) and Pīta varṇa (yellow colour).

- पाण्डुस्तु पीतभागार्द्धः केतकीधूलिसन्निभः ॥ (SKD)

Pāṇḍu resembles the color of pollen of Ketakī (Pandanus odoratissimus) flowers, which is yellowish white in colour.

- हरिणः पाण्डुः पाण्डुरीषत् पाण्डुस्तु धुसरः॥ (Amarakośa. 2/13)

Hariṇa (green/yellow), Pāṇḍu (yellowish white), Pāṇḍurīṣat (pale) and Dhusara (dusky white) are the synonyms of the word Pāṇḍu.

- हरितादिवर्णेभ्यः प्रधानेन वर्णेनोपलक्षितो रोगः पाण्डुरोगः। (Ca.Ca.Ci.16/1)
- पाण्डुत्वेनोपलक्षितो रोगः पाण्डुरोगः। (Ma.M.N.8/1)

The disease which is predominated by yellowish white discoloration is termed as Pāṇḍu roga.

- सर्वेषु चैतेष्विह पाण्डुभावो यतोऽधिकोऽतः खलु पाण्डुरोगः ॥ (Su.U.44/4)

Pāṇḍu bhāva (pallor) is the predominated feature in all varieties of the disease, hence called Pāṇḍu roga.

- ⊙ पाण्डुवर्णाधिक्यात् सर्वे एव पाण्डुरोगाः प्रोच्यन्त इति ॥ (Ḍa.Su.U.44/1)
- ⊙ ....पाण्डुत्वं तेषु चाधिकम्॥ यतोऽतः पाण्डुरित्युक्तः स रोगः.... ॥ (A.H.Ni.13/3-4)

The disease with Pāṇḍu varṇa adhikyam (excessive pallor) as a predominant feature is called Pāṇḍu roga.

Paryāya (synonyms)

- ⊙ स कामलापानकिपाण्डुरोगः कुम्भाह्वयो लाघर(व)कोऽलसाख्यः । (Su.U.44/6)

Kāmalā, Pānaki, Pāṇḍuroga, Kumbhāhva, Lāgharaka and Alasā are the synonyms of Pāṇḍu roga.

Sāmānya Nidāna (etiological factors)

- ⊙ व्यवायमम्लं लवणानि मद्यं मृदं दिवास्वप्नमतीव तीक्ष्णम् । निषेवमाणस्य विदूष्य रक्तं कुर्वन्ति दोषास्त्वचि पाण्डुभावम् ॥ (Su.U.44/3)

Excessive indulgence in Vyavāya (excessive coitus), intake of Amla (sour), Lavaṇa (saline), Madya (alcohol), Mṛd (clay) and Tīkṣṇa (pungent) and Divāsvapnam (excessive day sleep) aggravate the doṣas, which vitiates the Rakta and produces Pāṇḍu bhāva (pallor colour) of the Tvak (skin).

⊙ क्षाराम्ललवणात्युष्णविरुद्धासात्म्यभोजनात्। निष्पावमाषपिण्याकतिलतैलनिषेवणात्॥
विदग्धेऽन्ने दिवास्वप्नाद्व्यायामान्मैथुनात्तथा। प्रतिकर्मर्तुवैषम्याद्वेगानां च विधारणात्॥
कामचिन्ताभयक्रोधशोकोपहतचेतसः। समुदीर्णं यदा पित्तं हृदये समवस्थितम्॥
वायुना बलिना क्षिप्तं सम्प्राप्य धमनीर्दश। प्रपन्नं केवलं देहं त्वङ्मांसान्तरमाश्रितम्॥
प्रदूष्य कफवातासृक्त्वङ्मांसानि करोति तत्। पाण्डुहारिद्रहरितान् वर्णान् बहुविधांस्त्वचि॥
स पाण्डुरोग इत्युक्तः ...। (Ca.Ci.16/7-12)

- ◈ Excessive intake of Kṣāra (alkaline), Amla (sour), Lavaṇa (saline), Atyuṣṇa (very hot), Viruddha Asātmya bhojanāt (incompatible and unwholesome food);
- ◈ Use of Niṣpāva (Śimbi dhānya), Māṣa (black gram), Piṇyāka (oil cake) and Tila taila (sesame oil);
- ◈ Indulging in Divāsvapna (day sleep), Vyāyāma (exercise) and Maithuna (sexual intercourse) during Vidagdhe anne (indigestion of the food causing burning sensation);
- ◈ Pratikarma vaiṣamya (abnormal administration of Pañcakarma (five evacuative procedures),
- ◈ Ṛtu vaiṣamya (abnormality of seasons)

- ◈ Vega vidhāraṇāt (suppression of the natural urges);

If a person's mind afflicted with Kāma (passion), Cinta (worry), Bhaya (fear), Krodha (anger) and Śoka (grief) indulges in above etiological factors, it causes aggravation of Pitta located in the heart and this Pitta is propelled by powerful Vata into the Daśa dhamanīs (ten arteries attached to the heart) and spread to whole body and gets localized in between Tvaca (skin) and Maṃsa (muscles) and vitiates the Kapha, Vata, Rakta, Tvaca and Saṃprāpti and thereby produces various shades of colours like Pāṇḍu (pale), Hāridra (yellow) and Haritā (green) in the body. This condition is known as Pāṇḍu roga.

Sāmānya Saṃprāpti (general pathogenesis)

⊙ दोषाः पित्तप्रधानास्तु यस्य कुप्यन्ति धातुषु। शैथिल्यं तस्य धातूनां गौरवं चोपजायते॥
ततो वर्णबलस्नेहा ये चान्येऽप्योजसो गुणाः। व्रजन्ति क्षयमत्यर्थं दोषदूष्यप्रदूषणात्॥
सोऽल्परक्तोऽल्पमेदस्को निःसारः शिथिलेन्द्रियः। वैवर्ण्यं भजते, ...॥ (Ca.Ci.16/4-6)

Aggravated Pitta dominant three doṣas vitiates the dhātus and causes Śaithilyaṃ (sluggishness) and Gaurava (heaviness) in the dhātus and produces diminution of Varṇa (complexion), Bala (strength), Snehā (unctuousness) and Ojo guṇa (qualities of Ojas). By Atyarthaṃ doṣadūṣya pradūṣaṇāt (excessive vitiation of doṣas and dūṣyas), the Rakta (blood) and Medas (fatty tissue) gets diminished along with Niḥsāraḥ (loss of essence of dhātus), Śithilendriya (weak sense organs) and produces Vaivarṇya (abnormality of complexion).

Saṃprāpti Ghaṭaka

Doṣa	Pitta Pradhāna Tridoṣa
Dūṣya	Rasa, Rakta, Māṃsa, Meda, Tvak
Agni	Jaṭharāgni, Dhātvāgni māndhya
Āma	Jaṭharāgni, Dhātvāgni māndhya janya
Srotas	Rasa and Rakta vaha
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Sarva Śārīra
Udbhava sthāna	Āmāśaya
Vyakta sthāna	Tvak, Māṃsa, Nakha, Netra
Adhiṣṭhāna	Tvakmāṃsa antara

Rogamārga	Bāhya and Abhyantara
Svabhāva	Cirakāri

Pūrvarūpa (premonitory features)

- ⦿ ... तस्य लिङ्गं भविष्यतः। हृदयस्पन्दनं रौक्ष्यं स्वेदाभावः श्रमस्तथा॥ (Ca.Ci.16/12)
- ⦿ प्राग्रूपमस्य हृदयस्पन्दनं रूक्षता त्वचि। अरुचिः पीतमूत्रत्वं स्वेदाभावोऽल्पवह्निता॥ (A.H.Ni.13/8)

Premonitory symptoms of Pāṇḍu roga include Hṛdaya Spandanaṃ (palpitation of heart), Raukṣyaṃ (roughness), Svedābhāva (absence of sweat) and Śrama (tiredness). Ācārya Vāgbhaṭa added Aruci (anorexia), Pītamūtratvaṃ (yellowish urine) and Alpavahnitā (poor digestive fire).

- ⦿ त्वक्स्फोटनं ष्ठीवनगात्रसादौ मृद्भक्षणं प्रेक्षणकूटशोथः । विण्मूत्रपीतत्वमथाविपाको भविष्यतस्तस्य पुरःसराणि ॥ (Su.U.44/5)

Ācārya Suśruta added Tvak sphoṭana (cracks in the skin), Ṣṭhīvana (spitting), Gātrasāda (malaise), Mṛdbhakṣaṇaṃ (craving for eating clay), Prekṣeṇakūṭa śotha (edema around the eyelids), Viṇmūtra pītatva (yellowish discoloration of faeces and urine) and Avipāka (improper digestion).

Sāmānya Lakṣaṇās (general clinical features)

- ⦿ सम्भूतेऽस्मिन् भवेत् सर्वः कर्णक्ष्वेडी हतानलः। दुर्बलः सदनोऽन्नद्विट् श्रमभ्रमनिपीडितः॥
  गात्रशूलज्वरश्वासगौरवारुचिमान्नरः। मृदितैरिव गात्रैश्च पीडितोन्मथितैरिव॥
  शूनाक्षिकूटो हरितः शीर्णलोमा हतप्रभः। कोपनः शिशिरद्वेषी निद्रालुः ष्ठीवनोऽल्पवाक्॥
  पिण्डिकोद्वेष्टकट्यूरुपादरुक्सदनानि च। भवन्त्यारोहणायासैर्विशेषश्चास्य वक्ष्यते॥ (Ca.Ci.16/13-16)
  - ◈ Karṇa Kṣveḍī (tinnitus)
  - ◈ Hatānala (diminution of digestive power),
  - ◈ Durbala (debility),
  - ◈ Sadana (malaise)
  - ◈ Annadviṭ (aversion to food),
  - ◈ Śrama (exhaustion),
  - ◈ Bhrama (giddiness),
  - ◈ Gātra śūla (body ache),
  - ◈ Jvara (fever),
  - ◈ Śvāsa (dyspnoea),
  - ◈ Gaurava (heaviness),

  - ◈ Aruci (anorexia),
  - ◈ Mṛdita pīḍitonmathita gātra (feeling as if body is kneaded, pressed and churned),
  - ◈ Śūnākṣi kūṭa (swelling of orbital region),
  - ◈ Haritaḥ (green complexion),
  - ◈ Śīrṇa lomā (falling of skin hairs),
  - ◈ Hata prabhā (loss of luster),
  - ◈ Kopanaḥ (irritability),
  - ◈ Śiśira dveṣī (aversion to cold),
  - ◈ Nidrālu (over sleepiness)
  - ◈ Ṣṭhīvana (spitting),
  - ◈ Alpa vāk (reduced speech),
  - ◈ Piṇḍiko dveṣṭa (cramps in calf muscles) and
  - ◈ Kaṭyūrupādaruk sadanāni (pain and weakness in the waist, thighs and legs) especially by Arohaṇāyāsa (exertion by climbing) are the symptoms of Pāṇḍu roga.

Bheda (Classification of Pāṇḍu roga)

- ⦿ पाण्डुरोगाः स्मृताः पञ्च वातपित्तकफैस्त्रयः। चतुर्थः सन्निपातेन पञ्चमो भक्षणान्मृदः॥ (Ca.Ci.16/3)
- ⦿ पाण्ड्वामयोऽष्टार्धविधः प्रदिष्टः पृथक्समस्तैर्युगपच्च दोषैः । (Su.U.44/3)
- ⦿ स पञ्चधा पृथग्दोषैः समस्तैर्मृत्तिकादनात्। (A.H.Ni.13/7)
- ⦿ वातिकेः पैत्तिकश्चैव श्लैष्मिकः सान्निपातिकः। पञ्चमो रूक्षणः ....॥ (Hā.Sa.Tṛ.8/2)

Ācārya Caraka classified Pāṇḍu roga into five types namely Vātaja, Pittaja, Kaphaja, Sannipātaja and Mṛd bhakṣaṇaja (clay eating). Whereas, Ācārya Suśruta classified it into four types namely Vātaja, Pittaja, Kaphaja and Sannipātaja. Hārīta also classified Pāṇḍu roga into five types, Vātaja, Pittaja, Kaphaja, Sannipātaja and Rūkṣaṇa. Ācārya Hārīta mentioned Rūkṣaṇa for Mṛd bhakṣaṇajanya Pāṇḍu roga.

Pāṇḍu Bheda	Caraka	Vāgbhaṭa	Suśruta	Hārīta
Vātaja	+	+	+	+
Pittaja	+	+	+	+
Kaphaja	+	+	+	+
Sannipātaja	+	+	+	+
Mṛd bhakṣaṇaja	+	+	–	–
Rūkṣaṇa	–	–	–	+

Vātaja Pāṇḍu

- ⦿ आहारैरुपचारैश्च वातलैः कुपितोऽनिलः। जनयेत्कृष्णपाण्डुत्वं तथा रूक्षारुणाङ्गताम्॥
अङ्गमर्दं रुजं तोदं कम्पं पार्श्वशिरोरुजम्। वर्चःशोषास्यवैरस्यशोफानाहबलक्षयान्॥ (Ca.Ci.16/17-18)

- ⦿ ....अनिलात्तत्र गात्ररुक्तोदकम्पनम्। कृष्णरूक्षारुणसिरानखविण्मूत्रनेत्रता॥
शोफानाहास्यवैरस्यविट्शोषाः पार्श्वमूर्धरुक्। (A.H.Ni.13/9-10)

Intake of Vāta increasing diet and regimens aggravates Vāta and produces the symptoms of Vātaja Pāṇḍu such as Kṛṣṇa pāṇḍutvaṃ (blackish and pale yellowish discoloration), Rūkṣāruṇāṅgatāṃ (non-unctuousness and reddishness of the body), Aṅgamarda (body ache), Ruja (pain), Toda (pricking pain), Kampa (tremor), Pārṣva rujam (pain in flanks), Śira rujam (headache), Varca śoṣa (dried faeces), Āsyavairasya (bad taste in the mouth), Śopha (swelling), Ānāha (abdominal distention with constipation) and Balakṣaya (loss of strength).

- ⦿ कृष्णेक्षणं कृष्णसिरावनद्धं तद्वर्णविण्मूत्रनखाननं च । वातेन पाण्डुं मनुजं व्यवस्येद्युक्तं तथाऽन्यैस्तदुपद्रवैश्च ॥
(Su.U.44/7)

Suśruta says Kṛṣṇekṣaṇaṃ (blackish discoloration of eyes), Kṛṣṇa sirāvanaddham (blackish engorged veins), Tadvarṇa viṇmūtra nakhānanaṃ (blackish discoloration of faeces, urine, nails and face) and Anye upadrava (presence of other complication due to vitiation of Vāta) as symptoms of Vātaja Pāṇḍu.

Pittaja Pāṇḍu

- ⦿ पित्तलस्याचितं पित्तं यथोक्तैः स्वैः प्रकोपणैः। दूषयित्वा तु रक्तादीन् पाण्डुरोगाय कल्पते॥
स पीतो हरिताभो वा ज्वरदाहसमन्वितः। तृष्णामूर्च्छापिपासार्तः पीतमूत्रशकृन्नरः॥
स्वेदनः शीतकामश्च न चान्नमभिनन्दति। कटुकास्यो न चास्योष्णमुपशेतेऽम्लमेव च॥
उद्गारोऽम्लो विदाहश्च विदग्धेऽन्नेऽस्य जायते। दौर्गन्ध्यं भिन्नवर्चस्त्वं दौर्बल्यं तम एव च॥ (Ca.Ci.16/19-22)

- ⦿ पित्ताद्धरितपीताभसिरादित्वं ज्वरस्तमः॥ तृट्स्वेदमूर्च्छाशीतेच्छा दौर्गन्ध्यं कटुवक्त्रता। वर्चोभेदोऽम्लको दाहः......॥
(A.H.Ni.1310-11)

Intake of Pitta increasing diet and regimens aggravates Pitta which inturn affects the Rakta dhātu etc., and produces the symptoms of Pittaja Pāṇḍu such as Pītaharitābho (yellowish or greenish discoloration), Jvara (fever), Dāha (burning sensation), Tṛṣṇāmūrcchā (fainting caused by thirst), Pipāsā (thirst), Pītamūtrasakṛt (yellowish urine and faeces), Svedana (excessive perspiration), Śītakāma (desire for cold things), Annamabhinandati (does not relish food), Kaṭukāsya (pungent taste in the mouth), Na cāsysoṣṇamupaśeteamlameva (dislikes hot and sour things), Amlaudgāra (sour eructations), Vidāhaśca vidagdheanneasya (impaired digestion with burning sensation), Daurgandhyaṃ (foul smell), Bhinna varcas (diarrhoea), Daurbalya (weakness) and Tama (feeling of darkness).

- ⦿ पीतेक्षणं पीतसिरावनद्धं तद्वर्णविण्मूत्रनखाननं च । पित्तेन पाण्डुं मनुजं व्यवस्येद्युक्तं तथाऽन्यैस्तदुपद्रवैश्च ॥
(Su.U.44/8)

Suśruta says Pītekṣaṇaṃ (yellowish discoloration of eyes), Pītasirāvanaddhaṃ (yellowish engorged veins), Tadvarṇa viṇmūtra nakhānanaṃ (yellowish discoloration of faeces, urine, nails and face) and Anye upadrava (presence of other complication due to vitiation of Pitta) as symptoms of Pittaja Pāṇḍu.

Kaphaja Pāṇḍu

- ⦿ विवृद्धः श्लेष्मलैः श्लेष्मा पाण्डुरोगं स पूर्ववत्। करोति गौरवं तन्द्रां छर्दिं श्वेतावभासताम्॥
प्रसेकं लोमहर्षं च सादं मूर्च्छां भ्रमं क्लमम्। श्वासं कासं तथाऽऽलस्यमरुचिं वाक्स्वरग्रहम्॥
शुक्लमूत्राक्षिवर्चस्त्वं कटुरूक्षोष्णकामताम्। श्वयथुं मधुरास्यत्वमिति पाण्ड्वामयः कफात्॥ (Ca.Ci.16/23-25)

- ⦿ ......-कफाच्छुक्लसिरादिता॥ तन्द्रा लवणवक्त्रत्वं रोमहर्षः स्वरक्षयः। कासश्चछर्दिश्च ...... ॥ (A.H.N.13/11-12)

Due to intake of Kapha increasing diet and regimens Kapha aggravates and produces Pāṇḍu roga as said earlier with the symptoms like Gaurava (heaviness), Tandrā (drowsiness), Chardi (vomiting), Śvetāvabhāsa (whitish complexion), Praseka (salivation), Lomaharṣa (horripilation), Sāda (weakness), Mūrcchā (fainting), Bhrama (giddiness), Klama (tiredness), Śvāsa (dyspnoea), Kāsa (cough), Ālasya (laziness), Aruci (anorexia), Vāksvara grahaṃ (obstruction of speech and voice), Śuklamūtrākṣivarca (whitish coloration of the urine, eyes and feces), Kaṭurūkṣoṣṇa kāmata (desire for pungent, non-unctuous and hot articles), Śvayathu (swelling) and Madhurāsya (sweet taste in the mouth).

- ⦿ शुक्लेक्षणं शुक्लसिरावनद्धं तद्वर्णविण्मूत्रनखाननं च । कफेन पाण्डुं मनुजं व्यवस्येद्युक्तं तथाऽन्यैस्तदुपद्रवैश्च ॥
(Su.U.44/9)

Suśruta says Śuklekṣaṇaṃ (whitish discoloration of eyes), Śuklasirāvanaddhaṃ (whitish engorged veins), Tadvarṇa viṇmūtra nakhānanaṃ (whitish discoloration of faeces, urine, nails and face) and Anye upadrava (presence of other complication due to vitiation of Kapha) as symptoms of Kaphaja Pāṇḍu

Sannipātaja Pāṇḍu

- ⦿ सर्वान्नसेविनः सर्वे दुष्टा दोषास्त्रिदोषजम्। त्रिदोषलिङ्गं कुर्वन्ति पाण्डुरोगं सुदुःसहम्॥ (Ca.Ci.16/26)

- ⦿ ....निचयान्मिश्रलिङ्गोऽतिदुःसहः॥ (A.H.N.13/12)

Intake of all types of diet causes aggravation of all the three doṣas and produces Tridoṣaja Pāṇḍu having the clinical features of all the three doṣas and it is difficult to cure.

- ⦿ सर्वात्मके सर्वमिदं व्यवस्येद् वक्ष्यामि लिङ्गान्यथ कामलायाः । (Su.U.44/10)

In case of Sannipātaja Pāṇḍu, all the clinical features of the three doṣas are mixed together.

MṚDBHAKṢAṆAJA PĀṆḌU

- ⊙ मृत्तिकादनशीलस्य कुप्यत्यन्यतमो मलः। कषाया मारुतं, पित्तमूषरा, मधुरा कफम्॥
कोपयेन्मृद्रसादींश्च रौक्ष्याद्भुक्तं विरूक्षयेत् । पूरयत्यविपक्वैव स्रोतांसि निरुणद्धि च॥
इन्द्रियाणां बलं हत्वा तेजो वीर्यौजसी तथा। पाण्डुरोगं करोत्याशु बलवर्णाग्निनाशनम्॥
शूनगण्डाक्षिकूटभ्रूः शूनपान्नाभिमेहनः। क्रिमिकोष्ठोऽतिसार्येत मलं सासृक् कफान्वितम्॥ (Ca.Ci.16/27-30)

- ⊙ मृत्कषायाऽनिलं पित्तमूषरा मधुरा कफम्। दूषयित्वा रसादींश्च रौक्ष्याद्भुक्तं विरूक्ष्य च॥
स्रोतांस्यपक्वैवापूर्य कुर्याद्रुद्ध्वा च पूर्ववत्। पाण्डुरोगं ततः शूननाभिपादास्यमेहनः॥
पुरीषं कृमिमन्मुञ्चेद्भिन्नं सासृक्कफं नरः। (A.H.Ni.13/13-15)

Habitual intake of Mṛttikaā (clay) aggravates doṣas according to the taste of clay viz. Kasāya (astringent) aggravates Vāta, Ūṣarā (alkaline) aggravates Pitta and Madhura (sweet) aggravates Kapha doṣa. Besides this, it causes Raukṣyādbhuktaṃ virūkṣayet (unctuousness in the clay causes dryness in the Rasa dhātu and ingredients of food). This clay without being digested obstructs the Srotas of circulation and afflicts the Indriya bala (sensory strength), Tejas (luster), Vīrya (energy) and Ojas, and produces the Mṛdbhakṣaṇaja Pāṇḍu roga. Further it causes Balavarṇa agni nāśa (loss of strength, complexion and digestive power) and produces the symptoms such as Śūna gaṇḍākṣikūṭabhrū (swelling of the cheeks, orbital region and eyebrows) and Śūna pānnābhimehana (swelling in the legs, umbilical and pudendum region), Kṛmi koṣṭha (intestinal worms) and Atisāryeta malaṃ sāsṛk kaphānvitam (diarrhoea associated with blood and mucus).

Differentiate Doṣaja Pāṇḍu

Pāṇḍu	Vātaja Pāṇḍu	Pittaja Pāṇḍu	Kaphaja Pāṇḍu
Nidāna	Vātala Āhāra	Pittala Āhāra	Śleṣmala Āhāra
Varṇa	Kṛṣṇa Pāṇḍutva	Pīto haritābho vā	Śvetāvabhāda
Mūtrākṣivarca Varṇa	Kṛṣṇa	Pīta	Śukla
Āsya	Āsyavairasya	Kaṭukāsya	Madhurāsya
Anya Lakṣaṇa	Rūkṣāruṇa aṅgatam, Aṅgamarda, Ruja, Toda, Kampa, Pārśva rujam, Śira rujam, Varca śoṣa, Śopha, Ānāha, Balakṣaya	Jvara, Dāha, Truṣṇamūrccha, Pippāsa, Pītamūtrasakṛt, Svedana , Annamabhinandati, Uṣṇāmlānupaśayatā, Amlaudgāra, Vidāhasca vidagdheanneasya, Durgandha, Bhinna varcas, Daurbalya, Tama.	Gaurava, Tandrā, Chardi, Praṣeka, Lomaharṣa, Sāda Mūrccha, Bhrama, Klama, Śvāsa, Kāsa, Ālasya, Aruci, Vāksvara
Kāma	-	Śīta kāma	Kaṭurūkṣoṣṇā kāmata

Sādhyāsādhyata of Pāṇḍu roga (prognosis)

- ⊙ पाण्डुरोगश्चिरोत्पन्नः खरीभूतो न सिध्यति। कालप्रकर्षाच्छूनो ना यश्च पीतानि पश्यति॥
बद्धाल्पविट्कं सकफं हरितं योऽतिसार्यते। दीनः श्वेतातिदिग्धाङ्गश्छर्दिमूर्च्छातृषार्दितः॥
स नास्त्यसृक्क्षयाद्यश्च पाण्डुः श्वेतत्वमाप्नुयात्। इति पञ्चविधस्योक्तं पाण्डुरोगस्य लक्षणम्॥ (Ca.Ci.16/31-33)

Pāṇḍu roga which is Cirotpanna (chronic) and Kharībhūto (excessive non-unctuousness in the body) is not curable. Kalaprakarṣacchūno (edema of body parts for a long time in according with its chronicity), Pītāni paśyati (visualizing everything as yellow), Baddhālpa viṭ (passing hard faeces in less amount), Sakapha harita yoatisāryate (diarrhoea associated with mucus and green coloured faeces), Dīna (pathetic), Śvetātidigdhāṅga (looking extremely white as if being smeared), Chardi (vomiting), Mūrcchā (fainting), Tṛṣā (thirst) and Nāstyasṛk kṣayādyāśca pāṇḍu śvetatva (becomes whitish instead of pale colour due to extreme loss of blood and such patients never survives). These are the indicative signs of incurable Pāṇḍu roga of all types.

- ⊙ अन्तेषु शूनं परिहीनमध्यं म्लानं तथाऽन्तेषु च मध्यशूनम् । गुदे च शेफस्यथ मुष्कशूनं प्रताम्यमानं च विसञ्ज्ञकल्पम् ॥
विवर्जयेत् पाण्डुकिनं यशोऽर्थी तथाऽतिसारज्वरपीडितं च ॥ (Su.U.44/39-40)

The physician desiring fame, should reject the patients of Pāṇḍu roga who has Anteṣu śuna parihīna madhyaṃ (swelling in the extremities and emaciation in the middle part) or Mlānaṃ anteṣu ca madhyaśūna (swelling in the middle part and thinning of extremities), Guda śephasya muṣkaśūna (swelling of the anal region, penis and testes), Pratāmyamānaṃ (fainting) Visamjñakalpa (loss of consciousness), Atisāra (diarrhoea) and Jvara (fever).

Upadrava of Pāṇḍu roga (complications)

- ⊙ उपद्रवास्तेष्वरुचिः पिपासा छर्दिर्ज्वरो मूर्धरुजाऽग्निसादः । शोफस्तथा कण्ठगतोऽबलत्वं मूर्च्छा क्लमो हृद्यवपीडनं च ॥
(Su.U.44/13)

Aruci (anorexia), Pipāsa (thirst), Chardi (vomiting), Jvara (fever), Mūrdharuja (headache), Agnisāda (indigestion), Kaṇṭhagato Śopha (oedema in the throat region), Abalatvaṃ (loss of strength), Mūrcchā (fainting), Klamā (fatigue) and Hṛdya pīḍanaṃ (pain in the heart region) are the complication of Pāṇḍu roga.

In Contemporary science,

Pāṇḍu roga cannot be correlated with a particular disease. It can be a prodromal symptom, symptom, complication or a separate disease entity. Pāṇḍu roga is mentioned in both Santarpaṇa (over-nutrition) and Apatarpaṇa (under nutrition) Vyādhi, where there is Kapha doṣa predominance in the former and Pittavāta doṣa predominance in the latter. Hence, Santarpaṇottha Pāṇḍu roga can be correlated with Nutritional anaemia such as Iron deficiency anaemia and pernicious anaemia. Apatarpaṇottha Pāṇḍu roga can be correlated with Haemolytic anaemia; Leukaemia etc. (Please refer Topic 22)

Short Answer Questions

1. Explain Sāmānya Nidāna and Saṃprāpti of Pāṇḍu
2. Explain Pañca Pāṇḍu
3. Differentiate Doṣaja Pāṇḍu
4. Explain Mṛdbhakṣaṇaja Pāṇḍu
5. Describe Sādhyāsādhyata of Pāṇḍu roga

Long Answer Questions

1. Define Pāṇḍu and explain the Nidāna, Saṃprāpti, Bheda and Lakṣaṇās of Pāṇḍu

◆

TOPIC 21

Raktapitta

Introduction

Raktapitta is one of the diseases of Raktavaha srotas and occurs as the result of Pitta prakopa (aggravation of Pitta doṣa) and Rakta duṣṭi (vitiated Rakta dhātu). Mythologically Raktapitta is originated from Dakṣayajñadvaṃṣe (at the time of destruction of Daksha's Yajna) and Rudrakopāmārṣa (wrath and intolerance of Rudra) and appeared after Jvara (fever). Raktapitta is Mahāgada (major disorder), Mahāvega (spreads quickly) and Agnivat śīghrakāri (quickly acting like fire). Hence it should be treated promptly.

Nirūkti and Paribhāṣā

- रक्तदूषकं पित्तमिति । (SKD)
- रक्ते दूष्ये पित्तम् । (Ca.Ca.Ni.2/5)

The aggravated Pitta vitiating the Rakta is called Raktapitta.

- रक्तवत् पित्तं रक्तपित्तम्' इति । (Ca.Ca.Ni.2/5)
- रक्तं च पित्तं च रक्तपित्तमिति । (Ca.Ca.Ni.2/1-3)
- रक्तयुक्तं पित्तं रक्तपित्तम्' इति । (Ca.Ca.Ni.2/5)
- रक्तं च पित्तं चेति रक्तपित्तं । (Ma.M.N.9/1-3)

The Pitta attaining similar characteristics of Rakta is called Raktapitta.

- रक्तञ्च पित्तञ्च रक्तपित्तमिति । (SKD)

The aggravated Pitta producing the derangement of Rakta is called Raktapitta.

- अथ पित्तेन दुष्टं रक्तं रक्तपित्तमित्युच्यते । (Ma.M.N.9/1-3)

Pitta increases and aggravates the Rakta and this is called Raktapitta.

- संसर्गाल्लोहितप्रदूषणाल्लोहितगन्धवर्णानुविधानाच्च पित्तं लोहितपित्तमित्याचक्षते॥ (Ca.Ni.2/5)

Pitta is called Raktapitta or Lohitapitta because it comes in contact with Rakta and vitiates it and acquires its smell and colour.

- संयोगाद्दूषणात्तत्तु सामान्याद्गन्धवर्णयोः। रक्तस्य पित्तमाख्यातं रक्तपित्तं मनीषिभिः। (Ca.Ci.4/9)

Due to conjunction of Pitta with Rakta, Pitta vitiating Rakta dhātu and Pitta having similarity in Gandha (odor) and Varṇa (colour) with Rakta, it is called as Raktapitta.

- विनाऽभिघातात् स्फुटकारणाद् वा रक्तं दावेद् यत् प्रचुरं कुतश्चित्। तद् रक्तपित्तं भिषजो वदन्ति....॥ (Si.Ni.7/476)

Pracuraṃ rakta srava (abundant blood discharge) taking place in the body due to Vināabhighātāt (without any injury) or Vināsphuṭakāraṇāt (without any distinct cause), is generally called as Raktapitta by the physician.

- रक्तसंक्षोभणं पित्तं भूरि चेत् स्रावयेदसृक्। तिंह तद्रक्तपित्ताख्यं रोगं प्राञ्चः प्रचक्षते॥ (Si.Ni.7/477)

Ācāryas have illuminated Raktapitta as a disease in which the Pitta increases greatly, aggravates the Rakta and produces Bhūri cet rakta srāva (abundant blood discharge).

Nidāna

- यदा जन्तुर्यवकोद्दालककोरदूषप्रायाण्यन्नानि भुङ्क्ते, भृशोष्णतीक्ष्णमपि चान्यदन्नजातं निष्पावमाषकुलत्थसूपक्षारोप संहितं, दधिदधिमण्डोदश्वित्कट्वराम्लकाञ्जिकोपसेकं वा, वाराहमाहिषाविकमात्स्यगव्यपिशितं, पिण्याकपिण्डालुशुष्कशाकोपहितं, मूलकसर्षपलशुनकरञ्ज शिग्रुमधुशिग्रु (खडयूष) भूस्तृणसुमुखसुरसकुठेरक गण्डीरकालमालकपर्णा सक्षवकफणिज्झकोपदंशं, सुरासौवीरतुषोदकमैरेयमेदकमधूलकशुक्तकुवलबदराम्लप्रायानु पानं वा, पिष्टान्नोत्तरभूयिष्ठम्; उष्णाभितप्तो वाऽतिमात्रमतिवेलं वाऽऽमं पयः पिबति, पयसा समश्नाति रौहिणीकं, काणकपोतं वा सर्षपतैलक्षारसिद्धं, कुलत्थपिण्याकजाम्बवलकुचपक्वैः शौक्तिकैर्वा सह क्षीरं पिबत्युष्णाभितप्तः॥ (Ca.Ni.2/4)

Intake of Yavaka, Uddālaka, and Koradūṣa etc., cereals in excess quantities along with Uṣṇa (hot) and Tīkṣṇa (sharp) food items such as Niṣpāva, Māṣa, Kulattha and Kṣāra or with Dadhi, Dadhimaṇḍa, Udaśivat (buttermilk), Kaṭvara (sour buttermilk) or Kaṭvara mixed with Amlakāñjika; Meat of Vārāha, Māhiṣa, Āvika, Mātysa and Gavya with (vegetables) of Piṇyāka (oil cake), Piṇḍālu (a tuber) and Śuṣka śāka (dried pot herbs); Upadaṃśaṃ (anything eaten in addition for appetite) of Mūlaka, Sarṣapa, Laśūna, Karañja, Śigru, Madhuśigru or Khaḍayūṣa, Bhūstṛṇa, Sumukha, Surasa, Kuṭheraka, Gaṇḍīra, Kālamālaka, Parṇāsa, Kṣavaka and Phaṇijjhaka; Anupāna (after drink) of Surā, Sauvīra, Tuṣodaka, Maireya, Medaka, Madhūlaka, Śukta, sour preparations of Kuvala and Badara; or excessive intake of Piṣṭānna after taking meal; or excessive intake of unboiled milk frequently, especially after exposure to intense heat or intake of Rohiṇī along with milk; meat of Kāṇakapota cooked with Sarṣapa taila and Kṣāra; intake of milk cooked with either Kulattha, Piṇyāka, Jāmbu and Lakuca or else with Śauktikairvā after exposure to intense heat are the causative factors for Raktapitta.

- क्रोधशोकभयायासविरुद्धान्नातपानलान् । कट्वम्ललवणक्षारतीक्ष्णोष्णातिविदाहिनः ॥ (Su.U.45/3)
- तस्योष्णं तीक्ष्णमम्लं च कटूनि लवणानि च। घर्मश्चान्नविदाहश्च हेतुः पूर्वं निदर्शितः॥ (Ca.Ci.4/6)
- भृशोष्णतीक्ष्णकट्वम्ललवणादिविदाहिभिः। कोद्रवोद्दालकैश्चान्नैस्तद्युक्तैरतिसेवितैः। (A.H.Ni.3/1)

Krodha (anger), Śoka (grief), Bhaya (fear); Āyāsa (physical exertion), Viruddhānna (incompatible diet), Ātapānalān (exposure to sun and fire), excessive intake of Kaṭvamlalavaṇakṣāratīkṣṇoṣṇa (pungent, sour, saline, alkaline, sharp and hot substances) and Vidāhi (food substances which causes indigestion with burning sensation) are the causative factors for Raktapitta.

Samprāpti

- तस्यैवमाचरतः पित्तं प्रकोपमापद्यते, लोहितं च स्वप्रमाणमतिवर्तते।तस्मिन् प्रमाणातिवृत्ते पित्तं प्रकुपितं शरीरमनुसर्पद्यदेव यकृत्प्लीहप्रभवाणां लोहितवहानां च स्रोतसां लोहिताभिष्यन्दगुरूणि मुखान्यासाद्य प्रतिरुन्ध्यात् तदेव लोहितं दूषयति॥ (Ca.Ni.2/4)

By these causative factors, the Pitta gets aggravated and increases the quantity of Rakta in the body, where the aggravated Pitta enters into the circulation and reaches the Raktavahā sroto mūla i.e. Yakṛt and Plīha which are heavy due to flow of blood and obstructs their orifices by its Abhiṣyandi and Guru guṇa, thus the Pitta vitiates the Rakta and produces the disease Raktapitta.

- तैर्हेतुभिः समुत्क्लिष्टं पित्तं रक्तं प्रपद्यते। तद्योनित्वात् प्रपन्नं च वर्धते तत् प्रदूषयत्॥ तस्योष्मणा द्रवो धातुर्धातोर्धातोः प्रसिच्यते। स्विद्यतस्तेन संवृद्धिं भूयस्तदधिगच्छति॥ (Ca.Ci.4/7-8)

Due to the causative factors, Pitta gets aggravated and reaches Rakta. Because of their similarity in properties, the Pitta gets further aggravated. Due to the Ūṣmata (heat) of Pitta, there is an exudation of more liquids from the heated dhātus. These liquid forms of dhātus get mixed up with Pitta which is associated with Rakta, gets increased in quantity and produces the disease Raktapitta.

- नित्यमभ्यसतो दुष्टो रसः पित्तं प्रकोपयेत् । विदग्धं स्वगुणैः पित्तं विदहत्याशु शोणितम् ॥ (Su.U.45/4)

Due to causative factors, the Rasa gets vitiated which in turn aggravates the Pitta. The aggravated Pitta by its properties vitiates the Rakta and produces the disease Raktapitta.

Samprāpti Ghaṭaka

Doṣa	Pitta Pradhāna Tridoṣa
Dūṣya	Rasa and Rakta
Agni	Jaṭharāgni Dhātvāgni māndhya

Āma	Jaṭharāgni Dhātvāgni māndhya janya āma
Srotas	Rasa and Rakta vaha
Srotoduṣṭi	Atipravṛtti
Adhiṣṭhāna	Yakṛt and Plīha
Udbhava sthāna	Āmāśaya
Sañcāra sthāna	Rakta vaha
Vyakta sthāna	Ūrdhva adho mārga
Rogamārga	Bāhya and Abhyantara

Pūrvarūpa

- तस्येमानि पूर्वरूपाणि भवन्ति; तद्यथा- अनन्नाभिलाषः, भुक्तस्य विदाहः, शुक्ताम्लगन्धरस उद्गारः, छर्देरभीक्ष्णमागमनं, छर्दितस्य वीभत्सता, स्वरभेदो, गात्राणां सदनं, परिदाहः, मुखाद्धूमागम इव, लोहलोहितमत्स्यामगन्धित्वमिव चास्यस्य, रक्तहरितहारिद्रत्वमङ्गावयवशकृन्मूत्रस्वेद लालासिङ्घाणकास्यकर्णमलपिडकोलिकापिडकानाम्, अङ्गवेदना, लोहितनील पीतश्यावानामर्चिष्मतां च रूपाणां स्वप्ने दर्शनमभीक्ष्णमिति (लोहितपित्तपूर्वरूपाणि भवन्ति)॥ (Ca.Ni.2/6)
- शिरोगुरुत्वमरुचिः शीतेच्छा धूमकोऽम्लकः॥ छर्दिश्छर्दितबैभत्स्यंकासः श्वासो भ्रमः क्लमः। लोहलोहितमत्स्यामगन्धास्यत्वं स्वरक्षयः॥ रक्तहारिद्रहरितवर्णता नयनादिषु। नीललोहितपीतानां वर्णानामविवेचनम्॥ स्वप्ने तद्वर्णदर्शित्वं भवत्यस्मिन् भविष्यति। (A.H.Ni.3/4-7)

Anannābhilāṣa (aversion to food), Bhuktasya vidāha (improper digestion of food causing burning sensation), Śuktāmlagandha rasa udgāra (eructation having the smell and taste of Śuktamla (vinegar), Charderabhīkṣṇamāgamanaṃ (frequent vomiting), Charditasya bībhatsatā (unpleasant contents of vomitus), Svarabheda (hoarseness of voice), Gātra sada (malaise), Paridāha (burning sensation), Mukhāddhūmāgama (feeling as if smoke coming from the mouth), Loha lohita matsya āmagandhitvāsya (feeling of smell of metal, blood, fish and Āma in the mouth), Raktaharitahāridratvamaṅgāvayava śakṛtmūtra sveda lālāsiṅghaṇakāsya karṇamalapiḍakolikā (appearance of red, green or yellow colour in body parts, feces, urine, sweat, saliva, nasal secretion, excreta from mouth, ears and eyes); Piḍakāṇām (pimples), Aṅgavedana (body ache), Lohitanīlapītaśyāvanāmarciṣmatāṃ ca rūpaṇāṃ svapne darśanamabhīkṣṇamiti (frequent observation of red, blue, yellow, blackish and bright objects in dreams) are the premonitory features of Raktapitta.

- सदनं शीतकामित्वं कण्ठधूमायनं वमिः । लोहगन्धिश्च निःश्वासो भवत्यस्मिन् भविष्यति । । (Su.U.45/7-8)

Sadanaṃ (malaise), Śītakāmitvaṃ (desires for cold), Kaṇṭhadhūmāyanaṃ (feeling as if smokes are present in the throat), Vami (vomiting) and Lohagandhiśca niḥśvāso (metallic odour in the breath) are the premonitory features of Raktapitta.

Sāmānya Lakṣaṇās

- रक्तपित्तस्य लिगानि गात्राणां सदनं क्लमः। तृष्णा मोहस्तथा दाहो मनसः सम्भ्रमो भ्रमः॥
  शोणितच्छर्दनं वक्त्र लोहमत्स्यसगन्धता। रक्तातिसणं वाऽपि मार्गाभ्यां वा स्रवेदसृक्॥
  धमनीशिथिलत्वं च द्रुतस्पन्द शीतता। रक्तातिस्रवणात् क्वापि मृत्युर्वा बलसंज्ञयात्॥ (Si.Ni.7/496-498)

Gātrāṇāṃ sadanaṃ (weakness of the body), Klama (exhaustion), Tṛṣṇā (thirst), Moha (delusion), Dāha (burning sensation), Manasaḥ sambhramo (agitation of the mind), Bhrama (giddiness), Śoṇita chardi (blood vomiting), Vaktra lohamatsya sagandhatā (smell of metal and fish in the mouth), Raktātisaṇaṃ vāapi mārgābhyāṃ vā sravedasṛk (excessive bleeding from many passages), Dhamanī śithilatvaṃ (looseness of the arteries), Drutaspanda śītatā (body is cold to touch), Raktātisravaṇāt kvāpi mṛtyurvā (sometimes death may occur due to excessive bleeding) and Balasaṃjñayāt (loss of strength) are the general clinical features of Raktapitta.

Bheda and Lakṣaṇās

- मार्गौ पुनरस्य द्वौ ऊर्ध्वं, चाधश्च। तद्बहुश्लेष्मणि शरीरे श्लेष्मसंसर्गादूर्ध्वं प्रतिपद्यमानं कर्णनासिकानेत्रास्येभ्यः प्रच्यवते, बहुवाते तु शरीरे वातसंसर्गादधः प्रतिपद्यमानं मूत्रपुरीषमार्गाभ्यां प्रच्यवते, बहुश्लेष्मवाते तु शरीरे श्लेष्मवातसंसर्गाद्द्वावपि मार्गौ प्रतिपद्यते, तौ मार्गौ प्रतिपद्यमानं सर्वेभ्य एव यथोक्तेभ्यः खेभ्यः प्रच्यवते शरीरस्य॥ तत्र यदूर्ध्वभागं तत् साध्यं, विरेचनोपक्रमणीयत्वाद्बह्वौषधत्वाच्च; यदधोभागं तद्याप्यं, वमनोपक्रमणीयत्वादल्पौषधत्वाच्च; यदुभयभागं तदसाध्यं, वमनविरेचनायोगित्वादनौषधत्वाच्चेति॥ (Ca.Ni.2/8-9)
- ततः प्रवर्तते रक्तमूर्ध्वं चाधो द्विधाऽपि वा । आमाशयाद्व्रजेदूर्ध्वमधः पक्वाशयाद्व्रजेत् ॥
  विदग्धयोर्द्वयोश्चापि द्विधाभागं प्रवर्तते । केचित् सयकृतः प्लीह्नः प्रवदन्त्यसृजो गतिम् ॥ (Su.U.45/5-6)
- ऊर्ध्वं नासाक्षिकर्णास्यैर्मेढ्रयोनिगुदैरधः॥ कुपितं रोमकूपैश्च समस्तैस्तत्प्रवर्तते। (A.H.Ni.3/7-8)

Raktapitta has two pathways namely Ūrdhva (upwards) and Adhaḥ (downwards).

Ūrdhva Raktapitta : In the persons having predominance of Kapha in their body, the vitiated Pitta and Rakta associated with the Kapha move upwards from Āmāśaya and bleeding occurs from Karṇa (ears), Nāsikā (nose), Netra (eyes) and Āsya (mouth). It is Sādhya (curable), as it is Virecana yogitvāt (fits for the treatment of Virecana (purgation)), which is the best therapy to eliminate Pitta and also slightly effective in Kapha doṣa and is Bahu auṣadhatvāt (many drugs are available for the purpose). Madhura auṣadhas (sweet drugs) are useful along with Kaṣāya and Tikta rasa auṣadhas (astringent and bitter drugs).

Adhoga Raktapitta : In the persons having predominance of Vāta, the vitiated Pitta and Rakta associated with the Vāta moves downwards from Pakvāśaya and bleeding occurs from Mūtra mārga (urinary tract) and Purīṣa mārga (anal passage). Vāgbhaṭa stated that bleeding occurs through Meḍhra (penis), Yoni (vagina and genital tract) and Guda (rectum). It is

Yāpya (difficult to cure) because it is Vamanasya alpa yogitvāt (Vamana (emesis), which is less effective to eliminate Pitta) and is Alpauṣadhatvāt (lesser number of drugs are available for the purpose). Moreover Kaṣāya and Tikta auṣadhas (astringent and bitter drugs) are not suitable because it increases the Vāta doṣas. Only Madhura auṣadhas (sweet drugs) are useful to treat Adhoga Raktapitta, but its availability is very few.

Ubhaya Raktapitta : In the persons having predominance of Kapha and Vāta, the vitiated Pitta and Rakta associated with Kapha and Vāta moves upwards and downwards and bleeding occurs from all the orifices and pores of the body. Vāgbhaṭa stated that bleeding occurs even through Romakūpa (hair follicles). It is Asādhya (incurable), because of Vamanavirecana ayogitvāt (non applicability of both Virecana (purgation) and Vamana (emesis) and Anauṣadhatvāt (absence of suitable drugs).

Some Ācāryās stated that initially the Rakta moves from Yakṛt (liver) and Plīha (spleen), but comes out through Āmāśaya (stomach) and Pakvāśaya (large intestine).

◉ स्निग्धोष्णमुष्णरूक्षं च रक्तपित्तस्य कारणम्। अधोगस्योत्तरं प्रायः, पूर्वं स्यादूर्ध्वगस्य तु॥
ऊर्ध्वगं कफसंसृष्टमधोगं मारुतानुगम्। द्विमार्गं कफवाताभ्यामुभाभ्यामनुबध्यते॥ (Ca.Ci.4/23-24)

Raktapitta caused by Snigdhoṣṇa (unctuous and hot) articles gives rise to Ūrdhvaga Raktapitta and is associated with Kapha doṣa, whereas the disease Raktapitta caused by Uṣṇarūkṣaṃ (rough and hot properties) gives rise to Adhoga Raktapitta which is associated with Vāta doṣa. Ubhaya Raktapitta is associated with both Kapha and Vāta doṣas.

Doṣaja Bheda and Lakṣaṇās

◉ सान्द्रं सपाण्डु सस्नेहं पिच्छिलं च कफान्वितम्। श्यावारुणं सफेनं च तनु रूक्षं च वातिकम्॥
रक्तपित्तं कषायाभं कृष्णं गोमूत्रसन्निभम्। मेचकागारधूमाभमञ्जनाभं च पैत्तिकम्॥
संसृष्टलिङ्गं संसर्गात्त्रिलिङ्गं सान्निपातिकम्। (Ca.Ci.4/11-12)

◈ When aggravated Kapha is associated with Rakta, it becomes Sāndra (viscous), Sapāṇḍu (pale), Sasnehaṃ (unctuous) and Picchilaṃ (slimy); In Siddhānta nidānam, the special features of Kaphaja Raktapitta such as Kaphamiśrita Rakta (blood mixed with Kapha), Karapāda śītata (cold hand and feet) and Pratiśyāya (coryza) are described.

◈ When aggravated Vāta is associated with Rakta, it becomes Śyāva (greyish), Aruṇa (reddish), Saphena (frothy), Tanu (thin) and Rūkṣa (non- unctuous); In Siddhānta nidānam, the special features of Vātaja Raktapitta such as Saṃbhrama (giddiness), Kampa (tremors), Śiroruk (headache) and Śītakāmitva (desire for cold things) are described.

◈ When aggravated Pitta is associated with Rakta, it becomes Kaṣāyābhaṃ (red colour), Kṛṣṇa (blackish), Gomūtrasannibham (resembles cows urine), Mecaka (dark blue), Agāradhūma (house smoke) and Añjanābhaṃ (resembles black collyrium); In Siddhānta nidānam, the special features of Pittaja Raktapitta such

as Bhrama adhiko (severe giddiness), Dāho adhiko (severe burning sensation), Pittacharditve raktena (vomiting of Pitta with blood) and Raktātisāra (bloody diarrhoea) are described.

◈ If the disease Raktapitta occurs due to combination of two doṣas or all the three doṣas together, it manifests the features of respective doṣas involved.

Upadravā

◉ दौर्बल्यश्वासकासज्वरवमथुमदास्तन्द्रितदाहमूर्च्छा भुक्ते चान्ने विदाहस्त्वधृतिरपि सदा हृद्यतुल्या च पीडा ।
तृष्णा कण्ठस्य भेदः शिरसि च दवनं पूतिनिष्ठीवनं च द्वेषो भक्तेऽविपाको विरतिरपि रते रक्तपित्तोपसर्गाः ॥
(Su.U.45/9)

Daurbalya (debility), Śvāsa (dyspnoea), Kāsa (cough), Jvara (fever), Vamathu (vomiting), Mada (intoxication), Tandrā (drowsiness), Dāha (burning sensation), Mūrcchā (fainting), Bhukte cānne vidāha (burning sensation after taking food), Adhṛti (impatience) Sadā hṛdyatulyā ca pīḍa (constant pain similar to cardiac pain), Tṛṣṇā (thirst), Kaṇṭhasya bheda (pricking pain in the throat), Śirasi ca davanaṃ (pain in the head), Pūtiniṣṭhivanaṃ (putrid expectoration), Dveṣo bhakte (aversion to food), Avipāka (indigestion) and Viratirapirate (absence of desire for sexual intercourse) are the complication of Raktapitta.

◉ उपद्रवास्तु खलु दौर्बल्यारोचकाविपाकश्वासकासज्वरातीसारशोफशोषपाण्डुरोगाः स्वरभेदश्च॥ (Ca.Ni.2/7)

Daurbalya (debility), Arocaka (anorexia), Avipāka (indigestion), Śvāsa (dyspnoea), Kāsa (cough), Jvara (fever), Atisāra (diarrhea), Śopha (oedema), Śoṣa (emaciation), Pāṇḍu roga (anemia) and Svarabheda (hoarseness of voice) are the complication of Raktapitta.

Sādhyāsādhyata

◉ ऊर्ध्वं साध्यमधोयाप्यमसाध्यं युगपद्गतम् । (Su.U.45/7)

Ūrdhva Raktapitta is Sādhya (curable), Adhoga Raktapitta is Yāpya (difficult to cure) and Ubhaya Raktapitta is Asādhya (incurable).

◉ एकदोषानुगं साध्यं द्विदोषं याप्यमुच्यते॥ यत्त्रिदोषमसाध्यं तन्मन्दाग्नेरतिवेगवत्।
व्याधिभिः क्षीणदेहस्य वृद्धस्यानश्नतश्च यत्॥ (Ca.Ci.4/13-14)

Raktapitta associated with one doṣas is Sādhya (curable), that which is associated with two doṣas is Yāpya (difficult to cure) and that which is associated with three doṣas is Asādhya (incurable) and also it is Asādhya (incurable) when it manifests in a person having Mandāgni (subdued digestive fire), Ati vegavat (in severe form of disease), Vyādhibhi kṣīṇadehasya (who is emaciated due to the disease), Vṛddhasya (who is old) and Anaśnata (who is not able to eat).

Sādhyata of Raktapitta

- एकमार्गं बलवतो नातिवेगं नवोत्थितम्। रक्तपित्तं सुखे काले साध्यं स्यान्निरुपद्रवम्॥ (Ca.Ci.4/22)

Raktapitta afflicting through Eka mārga (one passage), in Balavān (strong person), Na ativegam (not in severe form), Navotthitam (recent manifestation), Sukhe kale (in favorable season) and Nirūpadrava (without any complications) is Sādhya (curable).

Asādhyata of Raktapitta

- एभ्यस्तु खलु हेतुभ्यः किञ्चित्साध्यं न सिध्यति॥ प्रेष्योपकरणाभावाद्दौरात्म्याद्वैद्यदोषतः। अकर्मतश्च साध्यत्वं कश्चिद्रोगोऽतिवर्तते॥ तत्रासाध्यत्वमेकं स्यात् साध्ययाप्यपरिक्रमात्। (Ca.Ni.2/21-22)

Even some Sādhya (curable) diseases become Asādhya (incurable) because of Preṣyābhāva (lack of attendants) and Upakaraṇābhāva (lack of equipments), Daurātmya (wickedness of the patient), Vaidya doṣata (fault of the physician) and Akarmata sādhyatvaṃ kaścidrogo ativartate (improper treatment to curable disease) which proceeds to the Yāpya avasthā (palliable stage) and then Asādhya avasthā (incurable stage).

- रक्तपित्तस्य विज्ञानमिदं तस्योपदिश्यते॥ यत् कृष्णमथवा नीलं यद्वा शक्रधनुष्प्रभम्। रक्तपित्तमसाध्यं तद्वाससो रञ्जनं च यत्॥ भृशं पूत्यतिमात्रं च सर्वोपद्रववच्च यत्। बलमांसक्षये यच्च तच्च रक्तमसिद्धिमत्॥ येन चोपहतो रक्तं रक्तपित्तेन मानवः। पश्येद्दृश्यं वियच्चापि तच्चासाध्यं न संशयः॥ (Ca.Ni.2/23-26)

Raktapitta which is Kṛṣṇa (black) or Nīla (blue) or Dhanuṣprabham (multiple colours like rainbow color), Vāsaso rañjanaṃ (even after washing the cloth, the blood stain persists), Bhṛśa pūtyatimātraṃ (frequent discharge of putrefied blood in excess), Sarvopadravavaccayat (associated with all complications) particularly in Balamāṃsa kṣaya (weak and emaciated patients) and Raktamasiddhimat paśyeddṛśyaṃ (if the patients sees all the visible objects and sky as red) is incurable.

- मांसप्रक्षालनाभं क्वथितमिव च यत् कर्दमाम्भोनिभं वा मेदःपूयास्रकल्पं यकृदिव यदि वा पक्वजम्बूफलाभम्। यत् कृष्णं यच्च नीलं भृशमतिकुणपं यत्र चोक्ता विकारास्तद्वर्ज्यं रक्तपित्तं सुरपतिधनुषा यच्च तुल्यं विभाति॥ (Su.U.45/10)

Raktapitta, which resembles Māṃsaprakṣālanābhaṃ (meat washings), Kvathitamiva (decoction) or Kardamāmbhonibhaṃ (muddy water) or Medaḥpūyāsrakalpa (mixed with fat or pus) or Yakṛdiva (liver tissue) or Pakvajambūphalābham (ripen Jambū fruit) and also, which resembles Kṛṣṇa (black) or Nīla (blue) and has Bhṛṣamatikūṇapaṃ (emits cadaveric smell), associated with Uktā vikārā (complications) and is of Surapatidhanuṣā (variegated colours like a rainbow), should be discarded from treatment as incurable.

- तत्रासाध्यं परित्याज्यं, याप्यं यत्नेन यापयेत्। साध्यं चावहितः सिद्धैर्भेषजैः साधयेद्भिषक्॥ (Ca.Ni.2/27)

The incurable one should be avoided, palliable one should be managed with efforts and the curable one should be treated successfully with suitable remedies by the physician.

In Contemporary science,

Raktapitta can be correlated with bleeding disorders. Bleeding disorders are characterised by prolonged bleeding time or clotting time as a result of disorders of coagulation factors, platelet abnormalities, vascular abnormalities or combination of all these factors.

Classification: Bleeding disorders are classified into inherited bleeding disorders and acquired bleeding disorders based on the causative factor of the disease.

- **Inherited bleeding disorders** are usually occurs due to defect in the coagulation factors or defects in the blood vessels right from birth. For example, coagulation disorders like Hemophilia A (factor-VIII deficiency), Hemophilia-B (factor-IX deficiency); connective tissue disorders like Ehlers-Danlos syndrome, osteogenesis imperfecta, Marfan syndrome; vascular disorders like Hereditary hemorrhagic telangiectasis etc.
- **Acquired bleeding disorders** are usually occurs due to trauma, inflammation, infection, neoplasm or drug-induced which leads to dysfunction or deficiency of coagulation factors and platelets. For example, Platelet disorders like Leukaemia, Aplastic anaemia, Idiopathic thrombocytopenic purpura; coagulation disorders like von Willebrand disease, Vitamin-K deficiency; vascular disorders like Vitamin-C deficiency.

Clinical features:

The most common clinical features of bleeding disorders are easily bruising, bleeding gum, epistaxis, malaena, haemoptysis, haematuria, mucosal bleeding, menorrhagia, petechiae, telangiectasia, ecchymosis, haemarthrosis, joint pain, abdominal pain, bloody diarrhoea and splenomegaly.

Diagnosis:

Diagnosis is based on the blood investigations which show decreased haemoglobin, red blood cells, white blood cells and platelets; prolonged prothrombin time; prolonged activated partial thromboplastin time; presence of antiplatelet antibodies; Bone marrow examination shows increased number of megakaryocytes.

MCQ's

1. Doṣa predominance in Raktapitta

a) Vāta b) Pitta

c) Kapha d) Sannipātaja

2. Raktapitta is
   - a) Mahāgada
   - b) Mahāvega
   - c) Agnivat śīghrakāri
   - d) All of the above

3. According to Suśruta, number of Raktapitta Pūrvarūpa
   - a) 5
   - b) 6
   - c) 7
   - d) 12

4. Pūrvarūpa of Raktapitta
   - a) Sadanam
   - b) Śītakāmitvam
   - c) Lohagandhiśca niḥśvāso
   - d) All of the above

5. Associated doṣas in the Adhoga Raktapitta
   - a) Vāta
   - b) Pitta
   - c) Kapha
   - d) Sannipāta

6. Karapāda śītata is the feature of
   - a) Vātaja Raktapitta
   - b) Pittaja Raktapitta
   - c) Kaphaja Raktapitta
   - d) None of the above

7. Ūrdhva Raktapitta is
   - a) Sādhya
   - b) Yāpya
   - c) Kṛcchra Sādhya
   - d) Asādhya

8. According to Caraka, number of Raktapitta Upadrava
   - a) 10
   - b) 11
   - c) 12
   - d) 15

9. What characterizes bleeding disorders
   - a) Rapid healing time
   - b) Prolonged bleeding time or clotting time
   - c) Increased platelet count
   - d) Enhanced blood flow

10. Which of the following is an example of an inherited bleeding disorder
    - a) Vitamin-C deficiency
    - b) Idiopathic thrombocytopenic purpura
    - c) Hemophilia A
    - d) Aplastic anaemia

11. What is the most common cause of acquired bleeding disorders
    - a) Genetic defects
    - b) Birth conditions
    - c) Trauma, inflammation, infection, neoplasm, or drug-induced factors
    - d) Overactive immune response

12. Which of the following is a vascular disorder that can lead to bleeding disorders
    - a) Hemophilia B
    - b) Hereditary hemorrhagic telangiectasia
    - c) Idiopathic thrombocytopenic purpura
    - d) Leukemia

13. What are the common laboratory findings in patients with bleeding disorders
    - a) Increased haemoglobin levels
    - b) Prolonged prothrombin time and activated partial thromboplastin time
    - c) Elevated white blood cell count
    - d) High platelet count

Answer Keys

1	b	2	d	3	a	4	d	5	a	6	c	7	a
8	b	9	b	10	c	11	c	12	b	13	b		

Short Answer Questions

1. Explain Sāmānya Nidāna and Saṃprāpti of Raktapitta
2. Explain the types of Raktapitta
3. Explain Adhoga Raktapitta
4. Explain Ūrdhvaga Raktapitta
5. Explain Sādhyāsādhyata of Raktapitta

Long Answer Questions

1. Define Raktapitta and explain the Nidāna, Saṃprāpti, Pūrvarūpa, Bheda and Viśiṣṭa Lakṣaṇās of Raktapitta

TOPIC 22

Hematopoietic Disease

Introduction

Hematopoietic diseases are a group of disorders that significantly impact the blood and blood-forming organs, such as bone marrow, spleen, and lymph node. These conditions can detrimentally affect the production and proper functioning of blood cells, which play a critical role in oxygen transport (red blood cells), immune response (white blood cells), and blood clotting (platelets). Some key types of hematopoietic diseases include anaemia, Thalassaemia, sickle cell anaemia, leukaemia, and thrombocytopenia.

ANAEMIA

Introduction

Anaemia is defined as an inadequate count of red blood cells (RBC) or haemoglobin concentration in blood, resulting in insufficient oxygen delivery to peripheral tissues. In 2019, WHO data indicated that roughly 29.9% of women aged 15-49 years and 39.8% of children aged 6-59 months were afflicted by anaemia. Within India, the prevalence of anaemia is notably elevated, with 67.1% of children aged 6-59 months affected, surpassing the global average of 39.8%.

According to the World Health Organization (WHO), haemoglobin (Hb) levels below 13.0 g/dL in adult males, below 12.0 g/dL in adult non-pregnant females, and below 11.0 g/dL in pregnant females signify the presence of anaemia.

Classification

Anaemia may be classified either on erythrocyte morphology or on their etiopathogenesis.

Based on Erythrocyte morphology:

The morphological classification relies on the correlation between red cell indices and the underlying cause of anaemia, with key measurements including red cell size (mean cell volume or MCV) and red cell haemoglobin concentrations (mean corpuscular haemoglobin (MCH) or mean corpuscular haemoglobin concentration (MCHC)). Anaemia can be categorized as macrocytic, normocytic, or microcytic based on red cell size (MCV), and as hypochromic or normochromic based on red cell haemoglobin concentration (MCH).

Microcytic hypochromic anaemia is characterized by reduced mean cell volume (MCV) and mean corpuscular haemoglobin (MCH), often seen in iron deficiency anaemia and Thalassaemia.

Macrocytic normochromic anaemia exhibits elevated MCV and normal MCH, typically associated with vitamin B12 deficiency anaemia and folate deficiency anaemia.

Macrocytic hypochromic anaemia, also known as dimorphic anaemia, manifests as varied red cell sizes and shapes, often due to mixed iron and vitamin deficiency.

Normocytic normochromic anaemia presents with normal MCV and MCH and is commonly associated to anaemia of chronic disease, renal failure, and haemolytic anaemia.

Based on etiopathogenesis

Anaemia can arise from blood loss, decreased erythropoiesis (hypoproliferative), or increased red blood cell destruction (haemolytic or hyperproliferative).

Blood loss can be acute or chronic blood loss, and increased RBC production occurs provided adequate nutrients are present. Causes of blood loss include trauma and gastrointestinal bleeding.

Hypo proliferative anaemia results from decreased RBC production due to acquired nutritional deficiencies or systemic diseases.

Haemolytic (hyper proliferative) anaemia is characterized by increased red blood cell destruction and elevated reticulocyte production. Causes include genetic conditions such as sickle cell anaemia, infections such as malaria, and certain autoimmune diseases.

Pathophysiology

Inadequate haemoglobin in the blood reduces oxygen-carrying capacity and results in compensatory physiological adaptations, including increased oxygen release from haemoglobin, increased blood flow to the tissues, preservation of blood volume, and redistribution of blood flow to sustain cerebral blood supply. Subsequently, tissue hypoxia ensues, impairing the functionality of the affected tissues. The extent of functional impairment varies depending on the oxygen demands of individual tissues. Tissues with high oxygen requirements, such as the heart, central nervous system, and skeletal muscle during exertion, are most affected by the clinical effects of anaemia.

Clinical features:

Symptoms of anaemia may vary depending on the underlying cause and severity. Common symptoms of anaemia are fatigue, weakness, pale skin, shortness of breath, dizziness or light-headedness, headaches, cold hands and feet, poor appetite, and weight loss. Physical examination reveals pallor, tachycardia, hypotension (orthostatic), systolic ejection murmur, splenomegaly, jaundice, petechiae, poor wound healing and lymphadenopathy.

Diagnosis:

Diagnosis is based on the clinical evaluation and blood test. The medical history helps to identify potential causes and risk factors for anaemia, such as family history of anaemia or blood disorders, previous blood transfusions or iron therapy, chronic diseases (e.g., kidney disease, rheumatoid arthritis), medications (e.g., NSAIDs, antibiotics), dietary habits and nutritional deficiencies, menstrual history (e.g., heavy menstrual bleeding), pregnancy or childbirth history, travel history (e.g., exposure to malaria) and social history (e.g., smoking, alcohol consumption). Physical examination helps to identify signs and symptoms of anaemia, such as pallor, tachycardia, etc.

Initial blood tests include a complete blood count (CBC), peripheral smear, and iron studies. The CBC measures haemoglobin (Hb), haematocrit, red blood cell count, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), and mean corpuscular haemoglobin concentration (MCHC). Peripheral smear examines blood cell morphology, while iron studies assess serum iron, total iron-binding capacity (TIBC), ferritin, and transferrin saturation. Additional tests may be necessary to determine the underlying cause, such as bone marrow biopsy, genetic testing, vitamin B12 and folate levels, reticulocyte count, erythropoietin level, haemoglobin electrophoresis, direct antiglobulin test (DAT), and indirect antiglobulin test (IAT). Specialized tests like flow cytometry, molecular testing, and imaging studies (e.g., CT, MRI) may also be used.

Differential diagnosis of Anaemia

The differential diagnosis of anaemia is broad, encompassing non-hematologic and hematologic causes.

Non-hematologic causes include chronic diseases like kidney disease, liver disease, HIV/AIDS, and rheumatoid arthritis; endocrine disorders such as hypothyroidism and adrenal insufficiency; nutritional deficiencies (vitamin B12, folate, iron, copper); and gastrointestinal disorders (celiac disease, Crohn's disease, ulcerative colitis).

Hematologic causes comprise red blood cell disorders, including iron deficiency anaemia, vitamin deficiency anaemia;, anaemia of chronic disease, sickle cell anaemia, Thalassaemia, and hereditary spherocytosis. White blood cell disorders, such as leukaemia and lymphoma, and platelet disorders like thrombocytopenia and thrombotic thrombocytopenic purpura, are also considered. Additionally, bone marrow failure syndromes (aplastic anaemia, myelodysplastic syndromes), medication-induced anaemia, anaemia of ageing, and pregnancy-related anaemia are potential causes.

NUTRITIONAL ANAEMIA

Introduction

Nutritional anaemia is a condition characterized by a deficiency in essential nutrients, particularly iron, vitamin B12, and folate, necessary to produce red blood cells. This deficiency leads to decreased haemoglobin levels, impaired oxygen delivery to tissues, and subsequent symptoms.

The National Health and Nutrition Examination Survey (NHANES) revealed that nutritional anaemia accounts for approximately 34% of cases in the elderly population, with varying rates reported in smaller studies. In India, an estimated 68.9% of children aged 1-4 years, 50.9% aged 5-9 years, and 65.1% aged 10-19 years suffer from nutritional anaemia, primarily due to poor dietary intake of iron, infections, genetic disorders, and cancers.

Nutritional anaemia is classified into

- Iron-deficiency anaemia
- Vitamin B12-deficiency anaemia
- Folate-deficiency anaemia
- Other nutritional anaemia

IRON DEFICIENCY ANAEMIA

Introduction

Iron Deficiency anaemia (IDA) is a microcytic and hypochromic anaemia characterized by a decrease in the number and size of red blood cells due to insufficient iron, leading to impaired oxygen delivery to tissues. According to the World Health Organization (WHO), Iron deficiency anaemia is the most common type of nutritional anaemia globally, affecting approximately 30% of the population, with a higher prevalence among children and women. At the same time, adult men are also susceptible, depending on their socioeconomic status and health conditions. The primary causes of Iron deficiency anaemia include

- Chronic blood loss (e.g., menstruation, gastrointestinal (GI) bleeding).
- Decreased dietary iron intake.
- Infections (e.g., hookworm).
- Increased iron requirement (e.g., pregnancy).
- Impaired iron absorption (e.g., celiac disease).

Pathophysiology:

Iron Deficiency anaemia (IDA) develops when the body's iron supply is inadequate for haemoglobin synthesis, resulting from increased blood loss, increased iron requirements, insufficient dietary intake, or decreased intestinal absorption. Initially, the body's stored iron, held in ferritin, is depleted, reducing iron availability for haemoglobin synthesis. Only after tissue stores are exhausted does the supply of iron to the bone marrow become insufficient, impairing haemoglobin formation and causing red blood cells to become microcytic (small) and hypochromic (pale). This gradual process progresses through stages: depletion of stored iron, reduced iron availability, impaired erythropoiesis, and ultimately, the development of microcytic, hypochromic anaemia.

Clinical features:

Iron deficiency anaemia may remain asymptomatic in most cases. However, as it progresses, general symptoms appear, including weakness, fatigue, dyspnoea on exertion, palpitations, loss of appetite, dyspepsia, constipation, and pallor of the skin, mucous membranes, and sclerae. Older patients may develop angina and congestive cardiac failure. Additional symptoms can include unusual dietary cravings, such as pica, and menorrhagia in iron-deficient women. Some patients may experience dysphagia due to the formation of webs at the postcricoid area, characteristic of Plummer-Vinson syndrome. Physical examination typically reveals generalized pallor, a sallow face, brittle grey hair, brittle nails, and koilonychia (spoon-shaped nails). Most cases also present with atrophic glossitis and angular stomatitis. Although less common, splenomegaly and hepatomegaly may occur. As the disease advances, oedema over-dependent parts and signs of congestive heart failure may develop.

Diagnosis:

The diagnosis of Iron deficiency anaemia is based on clinical evaluation and laboratory tests. A thorough medical history helps identify potential causes and risk factors, including dietary habits, menstrual history, chronic diseases, medications, and a family history of anaemia. Common symptoms include fatigue, weakness, shortness of breath, and headaches. Physical examination findings may include pallor, tachycardia, and koilonychia (spoon-shaped nails).

Laboratory tests including a Complete Blood Count (CBC) showing haemoglobin levels below 12 g/dL (women) or 13 g/dL (men), Mean Corpuscular Volume (MCV) below 80 fL, and Mean Corpuscular Haemoglobin (MCH) below 27 pg. Iron studies demonstrate serum iron below 50 µg/dL, serum ferritin below 15 ng/mL, and transferrin saturation below 16%. Additional tests may include stool occult blood, upper endoscopy, or colonoscopy to investigate gastrointestinal bleeding and imaging studies to evaluate underlying conditions.

VITAMIN B12-DEFICIENCY ANAEMIA

Introduction

Vitamin B12 deficiency anaemia is a megaloblastic and macrocytic anaemia characterized by insufficient levels of Vitamin B12, a critical nutrient for red blood cell production, leading to anaemia. This deficiency may arise from dietary insufficiency, malabsorption, increased physiological demand, or genetic disorders. In India, there is a notably high prevalence of vitamin B12 deficiency, affecting over 650 million individuals, with a particularly high incidence among women and children. This deficiency significantly contributes to the overall burden of anaemia in the country.

Pathophysiology:

Vitamin B12 deficiency anaemia arises when the body experiences inadequate levels of vitamin B12, a critical element for DNA synthesis, fatty acid metabolism, and nerve function. Normally, dietary vitamin B12 is assimilated in the ileum with the aid of intrinsic factor and subsequently conveyed to the liver for storage and dispersion. However, a scarcity of dietary intake or malabsorption, such as in cases of pernicious anaemia, gastric surgeries, or Crohn's disease, results in diminished vitamin B12 levels, thereby impeding DNA synthesis and inducing irregular erythrocyte morphology. This culminates in megaloblastic anaemia, manifested by the presence of large, immature red blood cells (megaloblasts) and heightened homocysteine levels. Furthermore, the deficiency impacts myelin synthesis, giving rise to neurological manifestations such as numbness, tingling, and cognitive impairments. Disrupted fatty acid metabolism escalates methylmalonic acid levels, further influencing energy production. Collectively, the cellular and molecular alterations encompass compromised DNA synthesis and repair, aberrant cell division and differentiation, disturbed mitochondrial function, and increased oxidative stress.

Clinical features:

Vitamin B12 deficiency anaemia presents with a wide range of clinical features, including fatigue, weakness, pale skin, jaundice, shortness of breath, and dizziness. Neurological symptoms, such as numbness, tingling, weakness, clumsiness, gait disturbances, ataxia, cognitive impairment, and mood changes, may also occur. Gastrointestinal symptoms include diarrhoea, constipation, abdominal pain, bloating, loss of taste, and weight loss. Haematological features comprise megaloblastic anaemia, macrocytosis (large red blood cells), anisocytosis (unequal red blood cell size), leucopenia (low white blood cell count), and thrombocytopenia (low platelet count).

The progression of Vitamin B12 deficiency anaemia can be categorized into four stages.

- **Stage 1, latent deficiency:** Characterized by mild decrease in Vitamin B12 levels without overt symptoms but with subclinical neurological changes.
- **Stage 2, mild deficiency:** Presents with moderate decrease in Vitamin B12 levels, mild anaemia, fatigue, and mild neurological symptoms.
- **Stage 3, moderate deficiency:** Marked by significant decrease in Vitamin B12 levels, megaloblastic anaemia, prominent neurological symptoms, and gastrointestinal symptoms.
- **Stage 4, severe deficiency:** Characterized by critically low Vitamin B12 levels, severe anaemia, weakness, advanced neurological symptoms, and increased risk of cardiovascular events.

If left untreated, Vitamin B12 deficiency anaemia can lead to permanent neurological damage, increased risk of cardiovascular disease, impaired cognitive function, and increased risk of infections.

Diagnosis:

Diagnosis is based on clinical evaluation, laboratory tests, and diagnostic criteria. Clinically, patients present with symptoms such as fatigue, weakness, pale skin, jaundice, shortness of breath, and neurological complaints. Laboratory tests include a complete blood count (CBC) to evaluate for anaemia, macrocytosis, and leucopenia, and a peripheral smear to confirm megaloblastic anaemia and macrocytosis. The definitive diagnostic measure involves the evaluation of serum Vitamin B12 levels (with levels below 200 pg/mL indicative of insufficiency), complemented by assessments of homocysteine and methylmalonic acid (MMA) levels to exclude folate deficiency.

Additional tests include intrinsic factor antibody to diagnose pernicious anaemia, gastrointestinal investigations (e.g., endoscopy, imaging) to identify underlying gastrointestinal disorders, and a folate level to rule out folate deficiency. Diagnostic criteria include low serum Vitamin B12, megaloblastic anaemia with macrocytosis, elevated homocysteine and/or methylmalonic acid, and response to Vitamin B12 supplementation. In certain instances, confirmatory assessments, such as the Schilling test (assessing Vitamin B12 absorption) and bone marrow biopsy (appraising megaloblastic alterations), may be necessary.

FOLATE-DEFICIENCY ANAEMIA

Folate-deficiency anaemia arises from insufficient folate (vitamin B9) in the body, leading to the impaired production of healthy red blood cells. Folate plays a crucial role in DNA synthesis, cell division, and the formation of red blood cells. This deficiency results in megaloblastic anaemia, characterized by the presence of large, immature red blood cells. Causes include inadequate dietary intake, malabsorption disorders, increased demand during pregnancy, and specific medications that disrupt folate metabolism.

Globally, approximately 2% of the population is affected by folate deficiency, with higher prevalence in regions with poor access to folate-rich foods. Certain groups, including pregnant women, infants, and individuals with malabsorption disorders, are at increased risk. In India, folate-deficiency anaemia is a significant public health concern, especially among vulnerable populations like women, children, and adolescents. According to the Comprehensive National Nutrition Survey, the prevalence of folate deficiency is 23% among preschool children, 28% among school-age children, and 37% among adolescents.

Pathophysiology:

Folate deficiency anaemia occurs when the body experiences an insufficient supply of folate, a vital nutrient essential for DNA synthesis and the production of red blood cells. Under normal circumstances, dietary folate is absorbed in the small intestine and transformed into tetrahydrofolate (THF), which provides methyl groups for DNA synthesis. However, inadequate dietary intake, malabsorption (e.g., celiac disease), or heightened physiological demand (e.g., pregnancy) result in diminished folate reserves. This deficiency hampers DNA synthesis, leading to aberrant erythropoiesis and culminating in megaloblastic anaemia typified by large, immature red blood cells. Folate insufficiency disrupts the methylation cycle, elevating homocysteine levels and impairing purine and pyrimidine synthesis. This disruption affects cell division, giving rise to anaemia, leucopenia, and thrombocytopenia. Furthermore, folate deficiency impedes neurotransmitter synthesis, contributing to neurological symptoms such as fatigue, weakness, irritability, and cognitive impairment.

Clinical features:

Folate deficiency anaemia manifests with a spectrum of clinical manifestations. Haematologically, it is characterized by megaloblastic anaemia, macrocytosis, anisocytosis, leucopenia, and thrombocytopenia. Clinical presentations include fatigue, weakness, lethargy, pallor, jaundice, dyspnoea, dizziness, headache, irritability, and anorexia. Neurological manifestations are prominent, featuring neuropathy (numbness, tingling), weakness, ataxia, cognitive impairment (memory loss, confusion), affective changes (depression, anxiety), and seizures (rare). Gastrointestinal symptoms encompass diarrhoea, constipation, abdominal pain, bloating, dysgeusia (loss of taste), and weight loss.

Untreated folate deficiency anaemia can lead to severe complications, including an elevated risk of cardiovascular disease, congenital abnormalities (e.g., neural tube defects), compromised cognitive function, and an increased susceptibility to infections. Pregnant women are particularly susceptible, with folate deficiency heightening the risk of premature labour, low birth weight, and foetal anomalies.

Diagnosis:

Diagnosis is based on clinical evaluation, laboratory tests including a complete blood count (CBC). Clinically, patients present with megaloblastic anaemia, macrocytosis, and neurological symptoms. Laboratory tests reveal low serum folate levels (<4 ng/mL), elevated homocysteine levels, and normal or elevated vitamin B12 levels. A complete blood count (CBC) shows anaemia, macrocytosis, and leucopenia, while a peripheral smear confirms megaloblastic changes.

Additional diagnostic measures may encompass a bone marrow biopsy to assess for megaloblastic changes, as well as gastrointestinal investigations (e.g., endoscopy, imaging) to ascertain underlying gastrointestinal disorders. Elevated serum homocysteine levels can be indicative of both folate deficiency and vitamin B12 deficiency, necessitating a critical differentiation between the two. The diagnosis is confirmed by a response to folate supplementation.

OTHER NUTRITIONAL ANAEMIA

Other nutritional anaemia's, aside from iron deficiency anaemia, vitamin B12 anaemia and folate deficiency anaemia, include, vitamin C deficiency anaemia, copper deficiency anaemia, zinc deficiency anaemia, and selenium deficiency anaemia. Other nutritional deficiencies contributing to anaemia include protein-energy malnutrition, vitamin A deficiency, vitamin E deficiency, magnesium deficiency, and manganese deficiency. Poor diet, malabsorption, increased physiological demand, chronic diseases, and certain medications can all contribute to nutritional anaemia.

- **Vitamin C deficiency anaemia** impairs iron absorption, causing fatigue, weakness, bleeding gums, and joint pain.
- **Copper deficiency anaemia,** resulting from inadequate copper intake or impaired absorption, causes fatigue, weakness, pale skin, and neurological symptoms.
- **Zinc deficiency anaemia,** often seen in growth retardation, impaired wound healing and immune dysfunction, is typically caused by inadequate zinc intake or impaired absorption.
- **Selenium deficiency anaemia,** characterized by cardiomyopathy, muscle weakness, and immune dysfunction, is linked to inadequate selenium intake or impaired absorption.
- **Protein-energy malnutrition** results in fatigue, weakness, poor growth in children, muscle wasting, and oedema due to insufficient intake of proteins and calories, leading to a lack of amino acids necessary for haemoglobin synthesis.
- **Vitamin A deficiency,** which plays a role in iron metabolism, causes symptoms such as night blindness, dry eyes and skin, increased susceptibility to infections, and fatigue.
- **Vitamin E deficiency** can lead to haemolytic anaemia, muscle weakness, and neurological issues such as poor coordination and nerve damage due to its role as an antioxidant protecting red blood cells from oxidative damage.
- **Magnesium deficiency** manifests as muscle cramps and spasms, fatigue, irritability, and irregular heart rhythms, as it is essential for proper enzyme function and energy production, indirectly affecting red blood cell production.
- **Manganese deficiency,** necessary for enzyme function and antioxidant defence, causes impaired growth and development, skeletal abnormalities, poor wound healing, fatigue, and weakness.

Differential diagnosis of Nutritional anaemia

The differential diagnosis of nutritional anaemia is extensive, necessitating meticulous consideration of various potential causes. In the case of iron deficiency anaemia, alternative diagnoses encompass anaemia of chronic disease, Thalassaemia , sideroblastic anaemia, and hemoglobinopathies. Distinguishing vitamin B12 deficiency anaemia from folate deficiency, pernicious anaemia, gastritis, small intestine bacterial overgrowth (SIBO), and pancreatic insufficiency is imperative.

Folate deficiency anaemia's differential diagnosis includes vitamin B12 deficiency, iron deficiency, anaemia of chronic disease, and hemoglobinopathies. Vitamin C deficiency anaemia may bear resemblance to iron deficiency, anaemia of chronic disease, scurvy, or hemorrhagic disorders. Copper deficiency anaemia shares similarities with iron deficiency, vitamin B12 deficiency, zinc deficiency, and Menkes disease.

Zinc deficiency anaemia's differential encompasses iron deficiency, copper deficiency, protein-energy malnutrition, and acrodermatitis enteropathica. Similarly, selenium deficiency anaemia may manifest in ways akin to cardiomyopathy, muscle weakness, thyroid disorders, or immune dysfunction. Proper differentiation of protein-energy malnutrition from marasmus, kwashiorkor, cachexia, and starvation is essential.

Vitamin A deficiency's differential diagnosis includes night blindness, dry eyes and skin, increased susceptibility to infections, and fatigue. Vitamin E deficiency may give rise to hemolytic anaemia, neurological disorders, muscle weakness, or ataxia. Manifestations of magnesium deficiency comprise muscle cramps, fatigue, irritability, or cardiac arrhythmias, while manganese deficiency manifests as impaired growth, skeletal abnormalities, poor wound healing, and fatigue.

Additional considerations encompass chronic diseases (e.g., kidney disease, HIV/AIDS), medications (e.g., antacids, proton pump inhibitors), malabsorption (e.g., celiac disease, Crohn's disease), and pregnancy and lactation. A comprehensive assessment of medical history, physical examination, laboratory results, and dietary habits is vital to ascertain the underlying cause of nutritional anaemia.

THALASSAEMIA (MEDITERRANEAN ANAEMIA)

Introduction

Thalassaemia is a heterogenous group of hereditary disorders characterized by impaired haemoglobin production, due to genetic mutations affecting alpha or beta globin chains. The condition manifests in two primary forms: alpha Thalassaemia and beta Thalassaemia. The severity of each type varies based on the specific genetic mutation and its consequential impact on haemoglobin production. Thalassaemia affects an estimated 1.7% of the global population, with a notably higher prevalence observed in Mediterranean, Middle Eastern, and South Asian demographics. In India, prevalence is estimated to range between 2.8% and 4.5%, with the highest incidence in the eastern and southern regions.

Pathophysiology

In Thalassaemia, mutated genes disrupt chain synthesis, leading to inadequate or dysfunctional haemoglobin. This results in ineffective erythropoiesis, reduced red blood cell count, and anaemia.

- **Alpha-Thalassaemia:** In alpha-Thalassaemia , the inadequate production of alpha-globin chains leads to an overabundance of beta chains, forming unstable tetramers such as haemoglobin H (HbH) and haemoglobin Bart's (Hb Bart's). These unstable haemoglobins impair the maturation of red blood cells, causing their premature destruction and consequent anaemia.
- **Beta-Thalassaemia:** In beta-Thalassaemia , the deficient production of beta-globin chains results in an excess of alpha chains that adhere to red blood cell membranes, inducing cell rigidity, fragmentation, and premature destruction.

The resultant anaemia activates compensatory mechanisms, including increased erythropoietin production, expansion of the bone marrow, and extramedullary haematopoiesis. Nevertheless, these mechanisms prove inadequate in addressing the severe anaemia, leading to symptoms such as fatigue, weakness, and shortness of breath.

Persistent haemolysis and transfusion therapy contribute to iron overload, potentially leading to cardiac and hepatic dysfunction, endocrine irregularities, and heightened susceptibility to infections. Furthermore, chronic anaemia and hypoxia associated with Thalassaemia give rise to expanded plasma volume, cardiac hypertrophy, and an increased risk of thrombosis.

Clinical Features:

The clinical manifestations of Thalassaemia are consistent but vary in severity based on the quantity of normal haemoglobin present. Common features encompass anaemia, jaundice, splenomegaly, hepatomegaly, bone alterations, heightened susceptibility to infections, and iron overload.

Alpha-Thalassaemia presents with varying severity based on the number of affected genes.

- **Silent Carrier:** Involvement of one gene, typically asymptomatic
- **Alpha Thalassaemia Trait:** Involvement of two genes, typically asymptomatic with mild microcytic anaemia and normal or slightly elevated HbA2.
- **Haemoglobin H (HbH) Disease:** Involvement of three genes, characterized by moderate to severe anaemia, fatigue, jaundice, splenomegaly, hepatomegaly, mild bone changes, and increased infection risk.
- **Hb Bart's Hydrops Fetalis:** Involvement of all four genes, typically resulting in intrauterine death or stillbirth, presenting with severe anaemia, congenital anomalies, cardiac failure, hepatomegaly, and splenomegaly.

Beta-Thalassaemia clinical phenotypes are categorized into three groups based on the extent of impaired beta globin production:

- **Thalassaemia Minor or Thalassaemia Trait:** A mild form, typically asymptomatic, characterized by mild microcytic anaemia and elevated HbA2.
- **Thalassaemia Intermedia:** Presents with moderate anaemia, jaundice, splenomegaly, hepatomegaly, mild to moderate bone changes, and increased infection risk..
- **Thalassaemia Major or Cooley's Anaemia:** Severe, marked by profound anaemia, jaundice, splenomegaly, hepatomegaly, severe bone changes, cardiac failure, growth retardation, pubertal delay, and significant iron overload.

Diagnosis:

Diagnosis is based on clinical evaluation, laboratory tests and genetic analysis. Clinical evaluation encompasses a review of medical history, a thorough physical examination, and an assessment of family history to identify indicative signs and symptoms such as anaemia, jaundice, splenomegaly, and bone deformities. Laboratory tests encompass a Complete

Blood Count (CBC), Peripheral Blood Smear, Haemoglobin Electrophoresis (HbE), High-Performance Liquid Chromatography (HPLC), and Capillary Electrophoresis to identify abnormal haemoglobin levels.

Confirmation of diagnosis and identification of specific mutations are achieved through DNA analysis and genetic testing. Prenatal diagnosis is feasible through procedures such as Chorionic Villus Sampling (CVS) and Amniocentesis to detect Thalassaemia in the foetus. Newborn screening programs utilize Guthrie card tests and HbE screening to detect Thalassaemia traits.

Diagnostic criteria for Alpha-Thalassaemia include reduced Mean Corpuscular Volume (MCV), elevated Hb Bart's or HbH, and reduced alpha-globin chain synthesis. Beta-Thalassaemia criteria include reduced MCV, elevated HbA2, and reduced beta-globin chain synthesis.

Differential diagnosis of Thalassaemia

Differential diagnosis of Thalassaemia includes various conditions with similar clinical and laboratory features. For Alpha-Thalassaemia, consider iron deficiency anaemia, anaemia of chronic disease, sickle cell disease, hemoglobinopathies (e.g., HbE, HbC), hereditary spherocytosis, pyruvate kinase deficiency, and glucose-6-phosphate dehydrogenase (G6PD) deficiency. For Beta-Thalassaemia, consider iron deficiency anaemia, vitamin B12 deficiency, folate deficiency, anaemia of chronic disease, sickle cell disease, hemoglobinopathies (e.g., HbS, HbC), and hereditary persistence of foetal haemoglobin (HPFH).

Other conditions to consider include congenital dyserythropoietic anaemia, Diamond-Blackfan anaemia, Fanconi anaemia, myelodysplastic syndromes, and bone marrow failure syndromes. Laboratory features such as haemoglobin electrophoresis, high-performance liquid chromatography (HPLC), capillary electrophoresis, DNA analysis, and mutation analysis help differentiate Thalassaemia.

SICKLE CELL ANAEMIA

Introduction

Sickle cell anaemia (SCA) is a genetic disorder caused by a mutation in the HBB (Haemoglobin Subunit Beta) gene, leading to abnormal haemoglobin production, which is the most common form of sickle cell disease (SCD). This mutation causes red blood cells to become sickle-shaped, rigid, and prone to destruction. Globally, sickle cell disease affects approximately 5% of the world's population. India bears the second-largest global burden of SCD, with over 1 million people affected, accounting for about 14.5% of global SCD newborns - roughly 42,000 cases annually.

Pathophysiology

Sickle Cell Anaemia (SCA) is caused by a mutation in the HBB gene, which leads to the production of abnormal haemoglobin known as haemoglobin S (HbS). This mutation causes a substitution of valine for glutamic acid at the sixth position of the beta-globin chain. Consequently, under low oxygen levels, HbS molecules aggregate to form elongated, inflexible rods within red blood cells, distorting their shape into a sickle form. These deformed cells are less pliant and prone to obstruction in small blood vessels, thus impeding blood flow. This vaso-occlusion leads to acute pain episodes, referred to as sickle cell crises, and can culminate in progressive organ damage.

The shortened lifespan of sickled cells results in chronic haemolytic anaemia as the body endeavours to replenish them at a sufficient pace. The continual breakdown of red blood cells induces jaundice and elevates the susceptibility to gallstones. Repetitive sickling and unsickling cycles inflict damage on blood vessel walls and instigate chronic inflammation, further exacerbating vaso-occlusion. The body endeavours to counteract chronic anaemia by augmenting red blood cell production in the bone marrow; however, this is often inadequate to match the rapid destruction of sickled cells, perpetuating persistent anaemia.

Clinical features:

Sickle Cell Anaemia manifests with a diverse array of clinical manifestations, exhibiting varying degrees of severity and frequency. Its primary symptom is the recurrent onset of intense pain, referred to as sickle cell crises, precipitated by the obstruction of blood flow and consequent tissue ischemia by sickled red blood cells. While these crises can impact any organ, they frequently involve the joints, back, and abdomen. A persistent feature of the condition is chronic haemolytic anaemia, characterized by pallor, jaundice, and fatigue. Patients also commonly experience recurrent infections, notably from encapsulated bacteria, due to splenic dysfunction.

Additional prevalent features encompass delayed growth and development in children, heightened susceptibility to stroke and other cardiovascular complications, organ damage, including kidney failure, liver dysfunction, and pulmonary hypertension, osteonecrosis due to impaired blood flow, priapism in males, and an elevated risk of vision loss due to retinopathy.

Furthermore, individuals affected by Sickle Cell Anaemia may display physical manifestations such as splenomegaly, hepatomegaly, cardiomegaly, and bone alterations, such as osteoporosis or deformities.

Diagnosis

Diagnosis is based on clinical evaluation, laboratory tests, and genetic analysis. Clinical evaluation plays a crucial role in identifying key signs and symptoms, including a family history of Sickle trait and recurrent episodes of severe pain, known as sickle cell crises. Additional clinical findings may include jaundice, pallor, splenomegaly, hepatomegaly, cardiomegaly, and bone alterations, such as osteoporosis or deformities.

Laboratory tests include peripheral blood smear examination, which reveals sickled red blood cells, while haemoglobin electrophoresis (HbE) and high-performance liquid chromatography (HPLC) identify and quantify abnormal haemoglobin (HbS). Additional tests, such as isoelectric focusing (IEF) and DNA analysis, detect the HBB gene mutation responsible for Sickle Cell Anaemia. Newborn screening programs, which involve collecting blood spots on filter paper for HbE or HPLC analysis, have become mandatory in many countries. Prenatal diagnosis is also possible through chorionic villus sampling (CVS) or amniocentesis. A diagnosis of Sickle Cell Anaemia is confirmed when HbS levels exceed 80%, HbA levels are absent or reduced, and clinical features consistent with Sickle Cell Anaemia are present.

Differential Diagnosis of Sickle Cell Anaemia

Sickle Cell Anaemia must be distinguished from other conditions manifesting similar symptoms or laboratory findings. Haematological disorders including Beta-Thalassemia Major, Beta-Thalassemia Intermedia, Haemoglobin C Disease, Haemoglobin E Disease, Hereditary Spherocytosis, and Pyruvate Kinase Deficiency may imitate SCA. Additionally, conditions such as Sickle Cell Trait (HbAS), Sickle-Beta Thalassaemia (HbS/β-thal), Chronic Haemolytic Anaemia, Autoimmune Haemolytic Anaemia, Glucose-6-Phosphate Dehydrogenase (G6PD) Deficiency, and Pyruvate Kinase Deficiency warrant consideration.

Non-haematological conditions, including vasculitis, osteoarthritis, rheumatoid arthritis, chronic pain syndromes, and infections such as osteomyelitis, may also present with analogous symptoms.

LEUKAEMIA

Introduction

Leukaemia is a malignant neoplasm of the hematopoietic tissues, characterized by the proliferation of abnormal white blood cells or their precursors in the bone marrow, blood, and other organs. Leukaemias are classified based on the predominant cell type involved (myeloid or lymphoid) and the natural history of the disease (acute or chronic). The main types of leukaemia are Acute Lymphoblastic Leukaemia (ALL), Acute Myeloid Leukaemia (AML), Chronic Lymphocytic Leukaemia (CLL), and Chronic Myeloid Leukaemia (CML).

Leukaemia accounts for approximately 3% of all cancer diagnoses worldwide, with an estimated global incidence of 454,000 new cases per year. In India, approximately 140,000 people live with leukaemia, primarily Acute Lymphoblastic Leukaemia (ALL), Acute Myeloid Leukaemia (AML), and Chronic Lymphocytic Leukaemia (CLL). Significant risk factors include genetic predisposition, radiation exposure, chemical exposure (e.g., benzene), viral infections (e.g., HTLV-1), family history, and previous cancer treatment.

Pathophysiology:

The pathophysiology of leukaemia involves genetic mutations, epigenetic changes, signalling pathway dysregulation, apoptosis resistance, and angiogenesis. Genetic mutations alter genes regulating cell growth, differentiation, and survival, while epigenetic changes affect DNA methylation and histone modification. Dysregulated signalling pathways, such as PI3K/AKT and MAPK/ERK, promote oncogenic activity. Apoptosis resistance inhibits programmed cell death, allowing abnormal cells to persist. Angiogenesis increases blood vessel formation, supporting leukemic cell growth.

Consequences of leukaemia include bone marrow failure, anaemia, thrombocytopenia, neutropenia, and increased susceptibility to infections. Leukaemia-specific pathophysiology varies: Acute Lymphoblastic Leukaemia (ALL) involves abnormal lymphoid cell proliferation; Acute Myeloid Leukaemia (AML), abnormal myeloid cell proliferation; Chronic Lymphocytic Leukaemia (CLL), accumulation of mature lymphocytes; and Chronic Myeloid Leukaemia (CML), abnormal myeloid cell proliferation with Philadelphia chromosome.

Clinical Features:

Leukaemia often presents with nonspecific symptoms, rendering early diagnosis challenging. Common manifestations encompass fatigue, weight loss, pallor, easy bruising and bleeding, recurrent infections, lymphadenopathy, bone and joint pain, abdominal discomfort, and anorexia. Specific symptoms exhibit variation contingent upon the leukaemia subtype.

- **Acute Lymphoblastic Leukaemia (ALL)** often involves lymphadenopathy, splenomegaly, hepatomegaly, and central nervous system manifestations such as headache and cognitive impairment..
- **Acute Myeloid Leukaemia (AML)** may cause gum infiltration and bleeding, skin lesions, respiratory distress, and cardiac complications.
- **Chronic Lymphocytic Leukaemia (CLL)** typically presents with lymphadenopathy, splenomegaly, fatigue, and weight loss.
- **Chronic Myeloid Leukaemia (CML)** often involves splenomegaly, hepatomegaly, weight loss, and fatigue.

Diagnosis

Diagnosis of leukaemia involves a combination of clinical evaluation, laboratory investigations, and imaging studies. Clinical assessment encompasses meticulous evaluation of patient history and symptoms, including fatigue, unexplained bruising, recurrent infections, and anaemia. Physical examination may reveal pertinent signs such as pallor, petechiae, ecchymoses, lymphadenopathy, and organomegaly.

Laboratory findings often demonstrate abnormal white blood cell counts, anaemia, and thrombocytopenia. A peripheral blood smear can unveil the presence of immature leukemic blast cells. Definitive diagnostic modalities encompass bone marrow aspiration and biopsy, elucidating the extent of marrow infiltration by leukemic cells. Immunophenotyping through flow cytometry facilitates classification of leukaemia subtypes by identifying specific cell markers. Genetic and molecular tests, including cytogenetic analysis and polymerase chain reaction (PCR), enable detection of chromosomal abnormalities and gene mutations associated with diverse leukaemia subtypes. Additional imaging studies, such as X-rays, CT scans, or MRIs, may be warranted to assess the involvement of other organ systems.

Differential diagnosis of leukaemia

The differential diagnosis of leukaemia encompasses consideration of various conditions that present similar clinical and laboratory features. These conditions encompass infections (e.g., mononucleosis, tuberculosis), lymphoma, lymphoproliferative disorders (e.g., lymphadenopathy), myeloproliferative neoplasms (e.g., polycythemia vera), myelodysplastic syndromes, and aplastic anaemia. Additional factors to consider include bone marrow failure syndromes (e.g., Fanconi anaemia), autoimmune disorders (e.g., rheumatoid arthritis), and medications that induce bone marrow suppression.

Specifically, Acute Lymphoblastic Leukaemia (ALL) may be misinterpreted as lymphoma, infectious mononucleosis, or aplastic anaemia. Acute Myeloid Leukaemia (AML) may resemble infections, myeloproliferative neoplasms, or myelodysplastic syndromes. Chronic Lymphocytic Leukaemia (CLL) may be mistaken for lymphoma or autoimmune disorders, while Chronic Myeloid Leukaemia (CML) may be confused with myeloproliferative neoplasms or bone marrow failure syndromes.

THROMBOCYTOPENIA

Introduction

Thrombocytopenia is characterized by an abnormally low number of platelets in the blood, specifically below 150,000/μl. Platelets are crucial for blood clotting, and their deficiency increases the risk of bleeding. However, spontaneous bleeding becomes evident only after a significant depletion of the platelet count to levels below 20,000/μl. Thrombocytopenia affects approximately 1.7% to 2.5% of the global population, with a higher occurrence among elderly individuals (over 65 years of age), females, and people with chronic illnesses. In India, the incidence ranges from 2.4 to 4.8 per 100,000 person-years, and the prevalence is 1.3% to 3.5% in the general population.

Pathophysiology

The pathophysiology of thrombocytopenia encompasses three primary mechanisms: decreased platelet production, increased platelet destruction, and sequestration of platelets in the spleen. Decreased platelet production arises from bone marrow disorders such as leukaemia, aplastic anaemia, or myelodysplastic syndromes, all of which compromise the bone marrow's capacity to generate platelets, resulting in an inadequate supply to meet physiological demands.

Increased platelet destruction often arises from immune-mediated processes in which antibodies target platelets, as seen in autoimmune diseases (e.g., immune thrombocytopenic purpura) or drug-induced thrombocytopenia (e.g., valproic acid, daptomycin, linezolid). Platelet consumption, exemplified by the rapid dissipation of platelets in disseminated intravascular coagulation (DIC) or thrombotic microangiopathy, also contributes to increased platelet destruction.

Platelet sequestration in the spleen diminishes their availability in the bloodstream. This is commonly observed in conditions such as liver cirrhosis and certain infections that lead to splenomegaly. Splenic sequestration confines platelets, impeding their circulation and contributing to thrombocytopenia.

Clinical Features:

Thrombocytopenia manifests with a spectrum of clinical manifestations, primarily associated with bleeding and haematological abnormalities. Prominent symptoms include petechiae (small, pinpoint bleeding spots), purpura (larger, purple skin patches), ecchymoses (bruises), and bleeding from the gums, nose, or gastrointestinal tract. Patients may also exhibit menorrhagia, haematuria, or bleeding from lacerations or trauma.

Additional clinical features may include fatigue, weakness, and shortness of breath due to anaemia, splenomegaly in instances of splenic sequestration, lymphadenopathy in the presence of underlying malignancy, pyrexia, rigors, or other signs of infection, as well as bone pain or tenderness in scenarios of bone marrow dysfunction.

In severe instances of thrombocytopenia (platelet count <10,000/μL), patients are vulnerable to life-threatening hemorrhagic complications, including intracranial haemorrhage, gastrointestinal bleeding, and hemorrhagic shock.

Diagnosis

Diagnosis is based on the clinical evaluation, laboratory investigations, and bone marrow aspiration. Clinically, patients commonly manifest with bleeding episodes, petechiae, purpura, or ecchymoses. A comprehensive medical history is elicited to identify potential causative factors such as medication usage, infections, or chronic diseases. Laboratory evaluations comprise a complete blood count (CBC) to determine platelet count, haemoglobin, hematocrit, and white blood cell count, in addition to a peripheral blood smear (PBS) for platelet morphology assessment.

Additional tests may be conducted, including bleeding time test to evaluate platelet function, and prothrombin time (PT) and activated partial thromboplastin time (aPTT) to rule out coagulopathy. Bone marrow aspiration and biopsy are conducted to evaluate bone marrow function and morphology. Specific assays, such as the von Willebrand factor (VWF) antigen test, are performed to identify underlying hereditary factors (e.g., von Willebrand disease). Anti-platelet antibody testing and Platelet-associated IgG (PAIgG) are utilized to rule out immune-mediated thrombocytopenia (ITP).

Differential diagnosis of Thrombocytopenia

The differential diagnosis of thrombocytopenia is extensive and necessitates a comprehensive assessment of clinical presentation, laboratory findings, and bone marrow results. It is imperative to differentiate pseudothrombocytopenia, such as EDTA-induced platelet clumping, from true thrombocytopenia. Various bone marrow disorders, including aplastic anaemia, myelodysplastic syndromes, leukemia, bone marrow fibrosis, and myeloproliferative neoplasms, can result in thrombocytopenia.

Immune-mediated thrombocytopenia, such as immune thrombocytopenia (ITP), systemic lupus erythematosus (SLE), and rheumatoid arthritis, should be taken into consideration. Infections, including bacterial sepsis, viral infections (e.g., HIV, hepatitis, EBV), fungal infections (e.g., candidemia), and parasitic infections (e.g., malaria), can also lead to thrombocytopenia.

Drug-induced thrombocytopenia, c caused by heparin, chemotherapy, antibiotics, and anticonvulsants, is another possibility. Chronic diseases such as liver cirrhosis, chronic kidney disease, diabetes mellitus, and hypersplenism can also contribute to thrombocytopenia. Hereditary thrombocytopenias, including von Willebrand disease, Bernard-Soulier syndrome, congenital amegakaryocytic thrombocytopenia, and thrombocytopenia-absent radius syndrome, merit consideration.

Coagulopathies, such as disseminated intravascular coagulation (DIC), thrombotic thrombocytopenic purpura (TTP), and haemolytic uremic syndrome (HUS), may also present with thrombocytopenia. Other causes, including pregnancy-related thrombocytopenia, nutritional deficiencies (e.g., vitamin B12, folate), and environmental toxins (e.g., pesticides), should not be disregarded.

MCQ's

1. Which type of anaemia is characterized by reduced mean cell volume (MCV) and mean corpuscular haemoglobin (MCH)

a) Normocytic normochromic anaemia b) Macrocytic normochromic anaemia
c) Microcytic hypochromic anaemia d) Macrocytic hypochromic anaemia

2. What is a common symptom of anaemia

a) Hypertension b) Excessive weight gain
c) Shortness of breath d) Decreased white blood cell count

3. What type of anaemia presents with normal MCV and MCH values

a) Microcytic hypochromic anaemia b) Normocytic normochromic anaemia
c) Macrocytic normochromic anaemia d) Macrocytic hypochromic anaemia

4. Which of the following laboratory tests is used to examine blood cell morphology

a) Complete Blood Count (CBC) b) Peripheral smear
c) Iron studies d) Bone marrow biopsy

5. Which nutritional deficiency is NOT commonly associated with nutritional anaemia

a) Iron b) Vitamin B12
c) Folate d) Vitamin D

6. What does a Complete Blood Count (CBC) typically show in a patient with Iron Deficiency Anaemia

a) Hemoglobin levels above 15 g/dL
b) Mean Corpuscular Volume (MCV) above 100 fL
c) Hemoglobin levels below 12 g/dL (women) or 13 g/dL (men)
d) Mean Corpuscular Hemoglobin (MCH) above 27 pg

7. Iron studies in patients with Iron Deficiency Anemia typically show serum iron levels below:

a) 70 μg/dL b) 60 μg/dL
c) 50 μg/dL d) 40 μg/dL

8. Which of the following is a characteristic physical examination finding in Iron Deficiency Anaemia

a) Bradycardia b) Brittle grey hair

c) Cyanosis d) Elevated blood pressure

9. Which symptom is associated with Plummer-Vinson syndrome in Iron Deficiency Anaemia

a) Jaundice b) Dysphagia

c) Night sweats d) Hyperpigmentation

10. What characterizes Vitamin B12 deficiency anaemia

a) Microcytic and normochromic anaemia b) Megaloblastic and macrocytic anaemia

c) Normocytic and normochromic anaemia d) Hypochromic and microcytic anaemia

11. What is a common clinical feature of Vitamin B12 deficiency anaemia

a) Hypertension b) Pale skin

c) Excessive weight gain d) Hyperpigmentation

12. Which symptom is NOT typically associated with folate-deficiency anaemia

a) Fatigue b) Jaundice

c) Weight gain d) Dyspnoea

13. Which of the following is a haematological feature of folate-deficiency anaemia

a) Hypochromia b) Macrocytosis

c) Spherocytosis d) Polycythemia

14. Which deficiency causes symptoms such as pale skin and neurological symptoms due to inadequate intake or impaired absorption

a) Vitamin C deficiency b) Copper deficiency

c) Zinc deficiency d) Selenium deficiency

15. Which of the following deficiencies is often seen in growth retardation, impaired wound healing, and immune dysfunction

a) Vitamin C deficiency b) Copper deficiency

c) Zinc deficiency d) Vitamin A deficiency

16. Selenium deficiency anaemia is characterized by which of the following symptoms

a) Night blindness b) Muscle weakness

c) Increased appetite d) Weight gain

17. Which deficiency manifests as muscle cramps, spasms, and irregular heart rhythms

a) Zinc deficiency b) Vitamin C deficiency

c) Manganese deficiency d) Magnesium deficiency

18. What characterizes Thalassaemia

a) Excessive production of hemoglobin

b) Impaired production of hemoglobin due to genetic mutations

c) Increased iron absorption

d) Decreased iron requirements

19. What is a common clinical feature of Beta-Thalassaemia Major?

a) Mild anaemia and normal HbA2

b) Severe anaemia, jaundice, and splenomegaly

c) Asymptomatic with normal red blood cells

d) Increased appetite and weight gain

20. Which diagnostic test is used to identify abnormal hemoglobin levels in Thalassaemia

a) Complete Blood Count (CBC) b) Peripheral Blood Smear

c) Haemoglobin Electrophoresis (HbE) d) Bone marrow biopsy

21. What genetic mutation causes Sickle Cell Anaemia (SCA)?

a) Mutation in the HBB gene b) Mutation in the HBA gene

c) Mutation in the HBC gene d) Mutation in the HBD gene

22. What complication arises from the continual breakdown of sickled red blood cells in SCA patients

a) Weight gain b) Gall stones

c) Hyperglycemia d) Hypotension

23. What is a common clinical manifestation of Leukaemia

a) Hypertension b) Increased appetite

c) Fatigue and weight loss d) Excessive hair growth

24. Which diagnostic test helps classify leukemia subtypes by identifying specific cell markers

a) Complete Blood Count (CBC)

b) Peripheral blood smear

c) Immunophenotyping through flow cytometry

d) Bone marrow biopsy

25. What symptom is a common clinical feature of thrombocytopenia

a) Excessive hair growth b) Petechiae

c) Weight gain d) Hypertension

Answer Keys

1	c	2	c	3	b	4	b	5	d	6	c	7	c	8	b	9	b
10	b	11	b	12	c	13	b	14	b	15	c	16	b	17	d	18	b
19	b	20	c	21	a	22	b	23	c	24	c	25	b				

Short Answer Questions

1. **Explain Anaemia**
2. **Explain Iron deficiency anaemia**
3. **Explain Sickle cell anaemia**
4. **Explain Thalassaemia**
5. **Explain Leukaemia**
6. **Explain Thrombocytopenia.**

◆

TOPIC 23

Hṛdroga

Introduction

Hṛdaya is an organ derived from Mātruja avayava (maternal part) and is formed from Śoṇitakapha prasāda (clear part of Rakta and Kapha). It is the seat of Ṣaḍaṅgam (six body parts), Aṅga (other parts than these six), Vijñāna (intellect), Indriya (sense faculties), Indriyartha pañcaka (five objects of senses), Ātma ca saguṇa (soul with its qualities), Cetascintyam (mind and objects of mind), Ojas and Prāṇavaha dhamanīs (life carrying arteries). Mahat and Artha are the synonyms of Hṛdaya. Hṛdaya is considered as one among the Trimarma (vital parts) and Daśaprāṇāyatana (ten seats of life). Hṛdaya looks like Adhomukha raktapadmakara (red lotus in an inverted position). Hṛdroga is a condition which causes discomfort of the heart as a result of vitiation of Rasa dhātu.

Nirūkti and Paribhāṣā

- हृदो रोगो हृद्रोगः। (Ma.M.N.29/2)
- हृदि रोगो हृद्रोग इति निरुक्तिः। (Āt.M.N.29/2)

The word Hṛdroga comprises of two words namely Hṛd and Roga. Hṛd means Hṛdaya (heart) and Roga means disease or pain. Hence the term Hṛdroga literally means disease afflicting the heart.

- हृदये बाधां हृद्रोगं तं प्रचक्षते ॥ (Su.U.43/4)

Hṛdaya bādhā (pain or discomfort in the heart) is called as Hṛdroga or in other words that which impairs the functions of the heart is known as Hṛdroga.

- हृद्रोगमिति वाच्ये यद्बाधाग्रहणं । (Ma.M.N.29/2)

That which interferes in the functions of heart presented with different type of pain or discomfort is called Hṛdroga.

- दूषयित्वा रसेदोषा विगुणा हृदिसंश्रिताः। पीडां तत्र प्रकुर्वन्ति हृद्रोगं तं प्रचक्षते॥ (Si.Yo.31/1)

The doṣas seated in Hṛdaya are aggravated and causes Rasa duṣṭi. This dūṣita rasa (vitiated Rasa dhātu) causes Pīḍā (pain) in the Hṛdaya (heart) which is called Hṛdroga.

Sāmānya Nidāna and Saṃprāpti

- व्यायामतीक्ष्णातिविरेकबस्तिचिन्ताभयत्रासगदातिचाराः। छर्द्यामसन्धारणकर्शनानि हृद्रोगकर्तॄणि तथाऽभिघातः॥ (Ca. Ci.26/77)

Vyāyāma (excessive physical exertion), Tīkṣṇātivirekabasti (sharp and excessive administration of purgation and enema therapies); Cintā (worry), Bhaya (fear), Trāsa (terror), Gadāticārā (improper management of diseases), Chardi (suppression of urge of vomiting), Āma doṣa, Sandhāraṇa (suppression of natural urges), Karśanāni (emaciation) and Abhighāta (trauma) are the causative factors of Hṛdroga.

- वेगाघातोष्णरूक्षान्नैरतिमात्रोपसेवितैः । विरुद्धाध्यशनाजीर्णैरसात्म्यैश्चापि(ति) भोजनैः ॥ (Su.U.43/3)

Vegāghāta (suppression of natural urges), Atimātra upasevita uṣṇa rūkṣānna (excessive intake of hot and non-unctuous foods), Viruddhāśana (incompatible foods), Adhyāśana (taking meal before the previous meal has been digested), Ajīrṇa (indigestion) and Asātmya bhojana (unwholesome food) are the causative factors of Hṛdroga.

- ... हृद्गदाः॥ तेषां गुल्मनिदानोक्तैः समुत्थानैश्च सम्भवः। (A.H.Ni.5/39)

Vāgbhaṭa stated that the disease Hṛdroga arise from the causative factors enumerated in Gulma nidāna.

- दूषयित्वा रसं दोषा विगुणा हृदयं गताः । कुर्वन्ति हृदये बाधां हृद्रोगं तं प्रचक्षते ॥ (Su.U.43/4)

Due to Nidāna sevana, the aggravated doṣas reaches the Hṛdaya and does Rasa dūṣaṇa (contamination of Rasa dhātu) which impairs the function of the heart and produces pain which is known as Hṛdroga.

Saṃprāpti Ghaṭaka

Doṣa	Tridoṣaja
Dūṣya	Rasa, Rakta
Agni	Jaṭharāgni and Dhātvāgni māndhya
Āma	Jaṭharāgni and Dhātvāgni māndhya janya
Srotas	Rasavaha, Mahās rotas
Srotoduṣṭi	Saṅga, Srotorodha

Udbhava sthāna	Āmapakvāśaya
Sañcāra sthāna	Rasavaha srotas and Daśa dhamanī
Vyakta sthāna	Hṛdaya
Adhiṣṭhāna	Hṛdaya
Rogamārga	Madhyama

Pūrvarūpa

- हृद्रोगे पूर्वरूपानभिधानमव्यक्तलक्षणानामेव पूर्वरूपत्वेनाभिमतत्वात्; ॥ (Ḍa.Su.U.43/3-4)

Avyakta lakṣaṇa (indistinct manifestations of the general symptoms of Hṛdroga just prior to the complete manifestation of the disease) is considered as the Pūrvarūpa (premonitory symptoms) of Hṛdroga.

Hṛdroga Bheda

- पञ्चैव हृदयामयाः। (Ca.Sū.17/6)
- स्मृताः पञ्च तु हृद्गदाः। (A.H.Ni.5/38)
- चतुर्विधः स दोषैः स्यात् कृमिभिश्च पृथक् पृथक् । लक्षणं तस्य वक्ष्यामि चिकित्सितमनन्तरम् ॥ (Su.U.43/5)

Caraka and Vāgbhaṭa classified Hṛdroga into five types namely Vātaja, Pittaja, Kaphaja, Sannipātaja and Kṛmija. While Suśruta classified into four types viz., Vātaja, Pittaja, Kaphaja and Kṛmija.

Sāmānya Lakṣaṇās of Hṛdroga

- वैवर्ण्यमूर्च्छाज्वरकासहिक्काश्वासास्यवैरस्यतृषाप्रमोहाः। छर्दिःकफोत्क्लेशरुजोऽरुचिश्चहृद्रोगजाःस्युर्विविधास्तथाऽन्ये॥ (Ca.Ci.26/78)

Vaivarṇya (discoloration), Mūrcchā (fainting), Jvara (fever), Kāsa (cough), Hikkā (hiccup), Śvāsa (dyspnoea), Āsyavairasya (bad taste in the mouth), Tṛṣā (thirst), Pramoha (delusion), Chardi (vomiting), Kaphotkleśa (excitation of Kapha or nausea), Ruja (pain), Aruci (anorexia) and Vividhāstathā anye (such other discomforts) are the general clinical features of Hṛdroga.

Vātaja Hṛdroga Nidāna and Lakṣaṇās

- शोकोपवासव्यायामरूक्षशुष्काल्पभोजनैः । वायुराविश्य हृदयं जनयत्युत्तमां रुजम्॥
  वेपथुर्वेष्टनं स्तम्भः प्रमोहः शून्यता दरः । हृदि वातातुरे रूपं जीर्णे चात्यर्थवेदना॥ (Ca.Sū.17/30-31)

Due to Śoka (grief), Upavāsa (fasting), Vyāyāma (excessive exercise) and Rūkṣa śuṣkālpa bhojana (intake of non-unctuous, dry and less quantity of food), the aggravated Vāta enters the Hṛdaya (heart) produces the disease Vātaja Hṛdroga and it is characterized by Uttama ruja (excruciating pain), Vepathu (trembling), Veṣṭanaṃ (cramps), Stambha (stiffness), Pramoha (delusion), Śūnyatā (sensation of emptiness in the heart), Daraḥ (tearing pain) and Jīrṇe cātyartha vedanā (pain aggravates after digestion of food).

- हृच्छून्यभावद्रवशोषभेदस्तम्भाः समोहाः पवनाद्विशेषः। (Ca.Ci.26/79)
- वातेन शूल्यतेऽत्यर्थं युद्यते स्फुटतीव च॥ भिद्यते शुष्यति स्तब्धं हृदयं शून्यता द्रवः।
  अकस्माद्दीनता शोको भयं शब्दासहिष्णुता॥ वेपथुर्वेष्टनं मोहः श्वासरोधोऽल्पनिद्रता। (A.H.Ni.5/39-41)

Hṛd śūnyabhāvat (sensation of emptiness in the heart), Hṛddrava (palpitation), Śoṣa (dryness), Bheda (piercing pain), Stambhā / Stabdha (stiffness) and Samohā (delusion) are the clinical features of Vātaja Hṛdroga.

Vāgbhaṭa added some features such as Atyartha yudhyate (severe pricking pain), Sphoṭyate (bursting pain), Bhidhyate (splitting or bursting pain), Śuṣyati (drying up pain), Akasmāddīnatā (abruptly weakness), Śoka (grief), Bhaya (fear), Śabdāsahiṣṇutā (aversion to sound), Vepathu (tremors), Veṣṭana (cramps or spasms), Śvāsarodha (obstructed breathing) and Alpanidratā (loss of sleep).

- आयम्यते मारुतजे हृदयं तुद्यते तथा । निर्मथ्यते दीर्यते च स्फोट्यते पाट्यतेऽपि च ॥ (Su.U.43/6)

Tudyate (pricking pain), Nirmathyate (churning pain), Dīryate (tearing pain), Sphoṭyate (bursting pain) and Pāṭyate (splitting pain) along with Āyamyate hṛdayaṃ (stretching sensation over the heart) are the clinical features of Vātaja Hṛdroga.

Pittaja Hṛdroga Nidāna and Lakṣaṇās

- उष्णाम्ललवणक्षारकटुकाजीर्णभोजनैः। मद्यक्रोधातपैश्चाशु हृदि पित्तं प्रकुप्यति॥
  हृद्दाहस्तिक्तता वक्त्रे तिक्ताम्लोद्गिरणं क्लमः। तृष्णा मूर्च्छा भ्रमः स्वेदः पित्तहृद्रोगलक्षणम्॥ (Ca.Sū.17/32-33)

Due to excessive intake of Uṣṇa (hot), Āmla (sour), Lavaṇa (Saline), Kṣāra (alkaline) and Kaṭu (pungent) food articles, Ajīrṇa bhojanaiḥ (eating during indigestion), Madya (alcohol), Krodha (anger) and Ātapa (exposure to sun), the Pitta in the Hṛdaya (heart) aggravated and produces the disease Pittaja Hṛdroga and it is characterized by Hṛddāha (burning sensation in the heart), Tiktā vakre (bitter taste in mouth), Tiktāmla udgāra (bitter and sour eructation), Klama (exhaustion), Tṛṣṇā (thirst), Mūrcchā (fainting), Bhrama (giddiness) and Sveda (perspiration).

- पित्तात्तमोदूयनदाहमोहाः सन्त्रासतापज्वरपीतभावाः॥ (Ca.Ci.26/79)
- पित्ता तृष्णा भ्रमो मूर्च्छा दाहः स्वेदोऽम्लकः क्लमः॥ छर्दनं चाम्लपित्तस्य धूमकः पीतता ज्वरः। (A.H.Ni.5/41-42)

Tama (feeling of darkness), Dūyana (heat in the body), Dāha (burning sensation), Mohā (delusion), Santrāsa (terror), Tāpa (heat sensation), Jvara (fever) and Pīta bhāvā (yellowishness of the body) are the clinical features of Pittaja Hṛdroga.

Vāgbhaṭa added some features such as Tṛṣṇā (thirst), Bhramā (giddiness), Mūrcchā (fainting), Sveda (perspiration), Amlaka (sourness), Klama (weakness), Chardanaṃ cāmlapittasya (vomiting of sour bile), and Dhūmaka (feeling of hot fumes).

- तृष्णोषादाहचोषाः स्युः पैत्तिके हृदयक्लमः । धूमायनं मूर्च्छा च स्वेदः शोषो मुखस्य च ॥ (Su.U.43/7)

Tṛṣṇā (thirst), Oṣā (increased temperature), Dāha (burning sensation), Coṣā (warmth), Hṛdaya klama (weakness of heart), Dhūmāyanaṃ (feeling of smoke in the mouth), Mūrcchā (fainting), Sveda (perspiration) and Mukha śoṣa (dryness of the mouth) are the clinical features of Pittaja Hṛdroga.

Kaphaja Hṛdroga Nidāna and Lakṣaṇās

- अत्यादानं गुरुस्निग्धमचिन्तनमचेष्टनम्। निद्रासुखं चाभ्यधिकं कफहृद्रोगकारणम्॥
  हृदयं कफहृद्रोगे सुप्तं स्तिमितभारिकम्। तन्द्रारुचिपरीतस्य भवत्यश्मावृतं यथा॥ (Ca.Sū.17/34-35)

Due to Atyādānaṃ (excessive eating), Gurusnigdha (intake of heavy and unctuous food substances), Acintanam (absence of mental worries), Aceṣṭanam (absence of physical activities) and Nidrā sukhaṃ cābhyadhikaṃ (excessive sleep) the aggravated Kapha enters the Hṛdaya (heart) produces the disease Kaphaja Hṛdroga and it is characterized by Hṛdaya Suptaṃ (inactivity of heart), Hṛdaya stimita (as if heart is covered with wet cloth) Hṛdaya bhārika (heaviness of heart as if pressed with a stone), Tandrā (drowsiness) and Aruci (anorexia) are the clinical features of Kaphaja Hṛdroga.

- स्तब्धं गुरु स्यात् स्तिमितं च मर्म कफात् प्रसेकज्वरकासतन्द्राः। (Ca.Ci.26/80)
- श्लेष्मणा हृदयं स्तब्धं भारिकं साश्मगर्भवत्॥ कासाग्निसादनिष्ठीवनिद्रालस्यारुचिज्वराः। (A.H.Ni.5/42-43)

Marma (heart) becomes Stabdha (stiffness), Guru (heavy) and Stimita (feeling as if covered with wet cloth); and also Praseka (salivation), Jvara (fever), Kāsa (cough) and Tandrā (drowsiness) are the clinical features of Kaphaja Hṛdroga.

Vāgbhaṭa added some features such as Hṛdaya (heart) becomes Stabdha (stiffens), Bhārikaṃ (heavy), and Sāśmagarbhavat (stone womb) associated with Agnisāda (weakness of digestion), Niṣṭhīva (spitting), Nidra (excessive sleep), Ālasya (lassitude), and Aruci (anorexia).

- गौरवं कफसंस्रावोऽरुचिः स्तम्भोऽग्निमार्दवम् । माधुर्यमपि चाऽऽस्यस्य बलासावतते हृदि ॥ (Su.U.43/8)

Gaurava (heaviness), Kaphasaṃsrāva (excessive secretion of mucus), Aruci (anorexia), Stambha (stiffness), Agnimārdava (impaired digestive fire) and Mādhuryāsyata (sweet taste in the mouth) are the clinical features of Kaphaja Hṛdroga.

Sannipātaja Hṛdroga Nidāna and Lakṣaṇās

- हेतुलक्षणसंसर्गादुच्यते सान्निपातिकः। (हृद्रोगः कष्टदः कष्टसाध्य उक्तो महर्षिभिः) (Ca.Sū.17/36)

Combination of all the causative factors and clinical features of all the three doṣas are present together and it is considered as the most troublesome and is difficult to cure.

- विद्यात्त्रिदोषं त्वपि सर्वलिङ्गं .... । (Ca.Ci.26/30)
- सर्वलिङ्गस्त्रिभिर्दोषैः.....। (A.H.Ni.5/43)

All the clinical features of all the three doṣas are manifested together.

Kṛmija Hṛdroga Nidāna and Lakṣaṇās

- त्रिदोषजे तु हृद्रोगे यो दुरात्मा निषेवते॥ तिलक्षीरगुडादीनि ग्रन्थिस्तस्योपजायते।
  मर्मैकदेशे सङ्क्लेदं रसश्चास्योपगच्छति॥ सङ्क्लेदात् क्रिमयश्चास्य भवन्त्युपहतात्मनः।
  मर्मैकदेशे ते जाताः सर्पन्तो भक्षयन्ति च॥ तुद्यमानं स हृदयं सूचीभिरिव मन्यते।
  छिद्यमानं यथा शस्त्रैर्जातकण्डूं महारुजम्॥ हृद्रोगं क्रिमिजं त्वेतैर्लिङ्गैर्बुद्ध्वा सुदारुणम्।
  त्वरेत जेतुं तं विद्वान् विकारं शीघ्रकारिणम्॥ (Ca.Sū.17/36-40)

If the Sannipātaja Hṛdroga person indulges in excessive intake of Tila (sesame), Kṣīra (milk), and Guḍa (jaggery) etc., Granthi (glandular structure) develops in one part of Marma (heart), there Rasa dhātu also attains moisture in one segment of the heart. Due to this moisture, Kṛmi (worms or parasites) develops in that part of the heart and it spreads to other parts of the heart and eats away the heart tissues and produces the disease Kṛmija Hṛdroga. It is characterized by Hṛdayaṃ Tudyamānaṃ śūcībhiriva manyate chidyamānaṃ yathā śastrai (feeling of pain as if pierced by needles or being cut by weapons), Kaṇḍū (itching sensation) and Mahā rujam (intense pain). It should be promptly diagnosed and managed immediately to control this disease because it is a very serious disorder which may lead to immediate death.

- .....तीव्रार्तितोदं कृमिजं सकण्डूम्॥ (Ca.Ci.26/30)
- ....कृमिभिः श्यावनेत्रता॥ तमःप्रवेशो हृल्लासः शोषः कण्डूः कफस्रुतिः। हृदयं प्रततं चात्र क्रकचेनेवदार्यते॥ चिकित्सेदामयं घोरं तं शीघ्रं शीघ्रकारिणम्। (A.H.Ni.5/43-45)

Tīvrārti todaṃ (severe discomfort and pricking pain) along with Sakaṇḍū (itching sensation) are the features of Kṛmija Hṛdroga.

Vāgbhaṭa added some features such as Śyāvanetratā (blackish discoloration of eyes), Tamaḥpraveśa (blackouts), Hṛllāsa (salivation), Śoṣa (emaciation), Kaṇḍu (itching), Kaphasruti (elimination of sputum), and Hṛdayaṃ pratataṃ cātra krakacenevadāryate (feels as if the heart is constantly being torn apart by a saw). He also mentioned that the disease spreads very quickly, so treatment should be started immediately.

- उत्क्लेशः ष्ठीवनं तोदः शूलो हृल्लासकस्तमः । अरुचिः श्यावनेत्रत्वं शोषश्च कृमिजे भवेत् ॥ (Su.U.43/9)

Utkleśa (nausea), Ṣṭhīvanaṃ (spitting), Toda (pricking pain), Śūla (colicky pain), Hṛllāsa (salivation), Tama (feeling of darkness), Aruci (anorexia), Śyāvanetratvaṃ (blackish discoloration of eyes) and Śoṣa (emaciation) are the features of Kṛmija Hṛdroga.

Upadravā of Hṛdroga

- भ्रमक्लमौ सादशोषौ ज्ञेयास्तेषामुपद्रवाः । कृमिजे कृमिजातीनां श्लैष्मिकाणां च ये मताः ॥ (Su.U.43/10)

Bhrama (giddiness), Klama (debility), Sāda (malaise) and Śoṣa (emaciation) are the complications of Hṛdroga. The complications seen in Kaphaja Kṛmiroga are also seen in the Kaphaja Hṛdroga.

Hṛdśūla

- कफपित्तावरुद्धस्तु मारुतो रसमूर्च्छितः ॥ हृदिस्थः कुरुते शूलमुच्छ्वासारोधकं परम्। स हृच्छूल इति ख्यातो रसमारुतसम्भवः ॥ (Su.U.42/131-132)

Vāta associated with the aggravated Rasa dhātu being obstructed by Kapha and Pitta, gets localized in the heart and produces Hṛcchūla (pain in the heart) which is characterized by Śūla (pain) and Ucchvāsārodhakam (difficulty in breathing). Thus the condition is predominantly produced by Rasa dhātu and Vāta doṣa.

In Contemporary science,

Hṛdroga can be correlated with cardiac disorders. Hṛdśūla is a pain predominant condition where there will be severe pain along with difficulty in breathing and it can be correlated with Angina pectoris due to presence of similar presentation. Vātaja Hṛdroga is also a pain predominant condition, so it can be correlated with Angina and due to defect in the Cala guṇa of Vāta, the heart may beat either very fast or slow leading to abnormal rhythm in the heartbeat resulting in Cardiac arrhythmia; Pittaja Hṛdroga can be correlated with inflammatory pathological conditions affecting the heart such as Myocarditis, Endocarditis and Pericarditis; Kaphaja Hṛdroga can be correlated with atherosclerotic changes (Dhamanī pratichāya) in the heart vessel conditions such as coronary artery disease including ischemic heart disease and also due to defect in the Sthira guṇa of Kapha, it may be lead to structural changes in the heart resulting in Left ventricular hypertrophy and Valvular disorders; Kṛmija Hṛdroga can be correlated with the diseases caused by different pathogens like bacteria, fungi and parasite such as infective endocarditis, chagas disease etc.

MCQ's

1. Adhomukha raktapadmakara is related to

a) Hṛdaya

b) Plīhā

c) Āmāśaya

d) Yakṛt

2. According to Caraka, Hṛdroga is of .... types

a) 4
b) 5
c) 6
d) 7

3. According to Suśruta, Hṛdroga is of .... types

a) 4
b) 5
c) 6
d) 7

4. According to Vāgbhaṭa, Hṛdroga Nidāna is similar to that of

a) Pāṇḍu Nidāna
b) Raktapitta Nidāna
c) Gulma Nidāna
d) Prameha Nidāna

5. आयम्यते हृदयं is the feature of

a) Vātaja Hṛdroga
b) Pittaja Hṛdroga
c) Kaphaja Hṛdroga
d) Sannipātaja Hṛdroga

6. हृदयक्लमः is the feature of

a) Vātaja Hṛdroga
b) Pittaja Hṛdroga
c) Kaphaja Hṛdroga
d) Sannipātaja Hṛdroga

7. साश्मगर्भवत् is the feature of

a) Vātaja Hṛdroga
b) Pittaja Hṛdroga
c) Kaphaja Hṛdroga
d) Sannipātaja Hṛdroga

8. तुद्यमानं स हृदयं सूचीभिरिव मन्यते is the feature of

a) Vātaja Hṛdroga
b) Pittaja Hṛdroga
c) Kaphaja Hṛdroga
d) Kṛmija Hṛdroga

9. Upadrava of Hṛdroga

a) Bhrama, Klama, Sāda and Śoṣa
b) Bhrama, Klama, Dāha and Tṛṣṇā
c) Sāda, Śoṣa, Dāha and Tṛṣṇā
d) None of the above

10. According to Vāgbhaṭa, which of the following is not a type of Hṛdroga

a) Sannipātaja
b) Kṛmīja
c) Raktaja
d) None of the above

Answer Keys

1	a	2	b	3	a	4	c	5	a	6	b	7	c	8	d	9	a	10	c

Short Answer Questions

1. Explain Sāmānya Nidāna and Saṃprāpti of Hṛdroga
2. Explain the Nidāna and Lakṣaṇās of Vātaja Hṛdroga
3. Explain the Nidāna and Lakṣaṇās of Pittaja Hṛdroga
4. Explain the Nidāna and Lakṣaṇās of Kaphaja Hṛdroga
5. Explain the Nidāna and Lakṣaṇās of Kṛmija Hṛdroga
6. Describe Hṛdśūla

◆

TOPIC 24

Śotha

Introduction

Śotha is a Tridoṣaja Vyādhi and comes under the diseases of Udakavaha srotas; it may occur as a separate disease or as a symptom in other diseases. Ācārya Caraka explained Śotha after Kṣatakṣīṇa because of their similarity in causative factor i.e. Marmopaghāta (injury to vital parts). Vāgbhaṭa explained Śotha along with Pāṇḍu roga, because Śotha is the Pūrvamupadravā (chief complication) of Pāṇḍu roga. Śopha, Śothaka and Śvayathu are the synonyms of Śotha.

Nirūkti and Paribhāṣā

- शोथः शवतीति । शु गतौ + वाहुलकात् । (SKD)
- शोफः शु गतौ । वाहुलकात् । (SKD)
- श्वयथुः श्वयतीति । श्वि गतिवृद्ध्योः । (SKD)

The word Śotha and Śopha are derived from the root 'Śu' which denotes "Gatau" which means to go and the word Śvayathu is derived from the root Śvi which denotes Gativṛddhoḥ which means to swell.

- उत्सेधलिङ्गं श्वयथुं करोति । (Ca.Ci.12/8)

Utsedha (elevation or protuberance) in any part of the body is called Śvayathu.

- सर्वमुत्सेधं शोथमाहुरित्यर्थः । (Ma.M.N.36/1-2)

All types of Utsedha (elevation or protuberance) in any part of the body are called Śotha.

- उत्सेधं संहतं शोफं तमाहुर्निचयादतः। (A.H.Ni.13/21)
- उत्सेधं संहतं शोथं तमाहुर्निचयादतः । (M.N.36/2)

That which is associated with Utsedha (elevation or protuberance) in any part of the body is called Śotha or Śopha.

Śvayathu bheda

- त्रयः शोथा भवन्ति वातपित्तश्लेष्मनिमित्ताः, ते पुनर्द्विविधा निजागन्तुभेदेन॥ (Ca.Sū.18/3)
- त्रिविधो निजश्च सर्वार्धगात्रावयवाश्रितत्वात्॥ (Ca.Ci.12/7)

Ācārya Caraka classified Śotha in to three types based on the predominance of doṣas i.e. Vātaja, Pittaja and Kaphaja. Again he classified into two types based on the causative factors i.e. Nija (endogenous) and Āgantuja (exogenous). Further he classified Nija Śvayathu (endogenous) into three types namely Sarva gātra (whole parts of the body), Ardha gātra (half parts of the body) and Avayavāśrita (any particular part of the body).

- दोषैः पृथग्द्वयैः सर्वैरभिघाताद्विषादपि। द्विधा वा निजमागन्तुं सर्वाङ्गैकाङ्गजं च तम्॥
  पृथून्नतग्रथिततताविशेषैश्च त्रिधा विदुः। (A.H.Ni.13/23-24)

Ācārya Vāgbhaṭa classified Śotha in to nine types based on the causes and symptoms i.e. Vātaja, Pittaja, Kaphaja, Vātapittaja, Pittakaphaja, Kaphavātaja, Sannipātaja, Abhighātaja and Viṣaja. Again he classified in to two types based on the causative factors i.e. Nija (endogenous) and Āgantuja (exogenous) and based on the Sthāna (location or place) i.e. Sarvāṅga (all parts of the body) and Ekāṅga (any one part of the body). Further he classified into three types based on the specific features i.e. Pṛthu (wide), Unnata (elevated) and Grathita (knotted).

- षड्विधोऽवयवसमुत्थः शोफोऽभिहितो लक्षणतः प्रतीकारतश्च; सर्वसरस्तु पञ्चविधः, तद्यथा- वातपित्तश्लेष्मसन्निपात विषनिमित्तः ॥ (Su.Ci.23/3)

Ācārya Suśruta classified Śotha in to six types based on the Avayava (regional) i.e. Vātaja, Pittaja, Kaphaja, Śoṇitaja, Sannipātaja and Āgantuja. Further he classified Sarvasara Śopha (generalized swelling) in to five types namely Vātaja, Pittaja, Kaphaja, Sannipātaja and Viṣaja.

Sāmānya Nidāna and Saṃprāpti

- सामान्यहेतुः शोफानां दोषजानां विशेषतः॥ व्याधिकर्मोपवासादिक्षीणस्य भजतो द्रुतम्। अतिमात्रमथान्यस्य गुर्वम्लस्निग्धशीतलम्॥ लवणक्षारतीक्ष्णोष्णशाकाम्बु स्वप्नजागरम्। मृद्ग्राम्यमांसवल्लूरमजीर्णश्रममैथुनम्॥ पदातेर्मार्गगमनं यानेन क्षोभिणाऽपि वा। श्वासकासातिसारार्शोजठरप्रदरज्वराः॥ विषूच्यलसकच्छर्दि गर्भवीसर्पपाण्डवः। अन्ये च मिथ्योपक्रान्तास्तैर्दोषा वक्षसि स्थिताः॥ ऊर्ध्वं शोफमधो बस्तौ मध्ये कुर्वन्ति मध्यगाः। सर्वाङ्गगाः सर्वगतं प्रत्यङ्गेषु तदाश्रयाः॥ (A.H.Ni.13/24-29)

Vyādhikarmopavāsādi kṣīṇa (those who become debilitated by diseases, evacuating therapies and fasting therapy etc.), Bhajate drutam atimātramathānyasya Gurvamla snigdhaśītalam (speedy and excessive intake of heavy, sour, unctuous and cold substances), Lavaṇakṣāratīkṣṇoṣṇa śākāmbu (saline, alkaline, sharp and heat producing vegetables and drinks), Svapnajāgaram (excessive sleep or keeping awake), Mṛdgrāmyamāṃsa (intake of mud and meat of domestic animals), Vallūra (dried meat), Ajirṇa śrama maithunam (copulation during indigestion and exertion), Padātermārga gamana yānena kṣobhiṇāpi vā (walking long distance or riding on animals or vehicles); as a effect of diseases such as Śvāsa, Kāsa, Atisāra, Arśas, Jaṭhara, Pradara, Jvara, Viṣuci, Alasaka, Chardi, Garbha, Visarpa Pāṇḍuroga and such other diseases that are improperly treated.

By these above said causative factors, the doṣas aggravated and localized in the chest produces Śopha in the upper parts of the body; if it is localized in the Basti (bladder), produces Śopha in the lower parts of the body; if it is localized in Madhya bhāga (middle parts), produces Śopha in the middle parts of the body; if localized in Sarvāṅga (all over the body), then it produces Śopha in all over the body and if localized in Ekāṅga (any one part), produces Śopha in that part only.

Nija Śotha Nidāna

- शुद्ध्यामयाभक्तकृशाबलानां क्षाराम्लतीक्ष्णोष्णगुरूपसेवा। दध्याममृच्छाकविरोधि दुष्टगरोपसृष्टान्ननिषेवणं च॥ अर्शांस्यचेष्टा न च देहशुद्धिर्मर्मोपघातो विषमा प्रसूतिः। मिथ्योपचारः प्रतिकर्मणां च निजस्य हेतुः श्वयथोः प्रदिष्टः॥ (Ca.Ci.12/5-6)

The one who became Kṛśa (emaciated) and Abala (weak) due to Śuddhaya (evacuative therapies), Āmaya (diseases), Abhakta (less or no intake of food) and Kṣārāmlatīkṣṇoṣṇagurupaseva (excessive use of alkali, sour, sharp, hot and heavy food substances); excessive intake Dadhi (curd), Āma (uncooked food substances) Mṛt (soil), Śāka (leafy vegetables), Virodhi (contradictory food substances), Duṣṭa (spoiled or stale food items), Garopasṛṣṭānna (poisoned food); Arśas (suffering from piles), Aceṣṭā (absence of physical activities), Na ca dehaśuddhi (not performing evacuative therapies), Marmopaghāta (injury to the vital parts), Viṣamā prasūti (abnormal delivery), Mithyopacāraḥ pratikarmaṇām (improper application and complication of evacuative therapies) are the causative factors of Nija Śotha.

Āgantu Śotha Nidāna

- बाह्यास्त्वचो दूषयिताऽभिघातः काष्ठाश्मशस्त्राग्निविषायसाद्यैः । आगन्तुहेतुः.... । (Ca.Ci.12/7)

The causative factors of Āgantu Śotha are Kāṣṭhāśmaśastrāgniviṣāyasādyaiḥ Abhighāta (injury with wood, stone, instruments, fire, poison and iron etc.) produces Bāhya tvacā dūṣāyitva (morbidity of the external skin) resulting in Śotha.

- बाह्याः सिराः प्राप्य यदा कफासृक्पित्तानि सन्दूषयतीह वायुः। तैर्बद्धमार्गः स तदा विसर्पन्नुत्सेधलिङ्गं श्वयथुं करोति॥ उरःस्थितैरूर्ध्वमधस्तुवायोःस्थानस्थितैर्मध्यगतैस्तुमध्ये।सर्वाङ्गगःसर्वगतैःक्वचित्स्थैर्दोषैःक्वचित्स्याच्छ्वयथुस्तदाख्यः॥ (Ca.Ci.12/8-9)

By these causative factors, Vāta gets vitiated and it enters the Bāhyaḥ siraḥ (external vessel) and afflicts the Kapha, Rakta and Pitta doṣas located at the site resulting in obstruction of passage of Vāta and then while spreading it produces the Utsedha (protuberance) which is the Pratyātma lakṣaṇa (cardinal symptom) of Śotha.

If the aggravated doṣas are localized in the chest, then produces Śopha in the upper parts of the body; if it is localized in the Vāta sthāna (Pakvāśaya), produces Śopha in the lower parts of the body; if it is localized in Madhya bhāga (middle parts), produces Śopha in the middle parts of the body; if localized in Sarvāṅga (all over the body), then it produces Śopha all over the body and if localized in Ekāṅga (any one part), Śopha of that part takes place and is named as per the location that it affects..

Samprāpti Ghaṭaka of Śotha:

Doṣa	Tridoṣa
Dūṣya	Rasa, Rakta
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Agni māndhya janya Āma
Srotas	Rasa, Rakta, Udaka and Sveda vaha
Srotoduṣṭi	Saṅga, Vimārga gamana
Sañcāra sthāna	Sarva śārīra
Udbhava sthāna	Āmapakvāśaya
Vyakta sthāna	Tvak
Adhiṣṭhāna	Tvakmāṃsāśraya
Rogamārga	Bāhya

Pūrvarūpa of Śotha

- ऊष्मा तथा स्याद्दवथुः सिराणामायाम इत्येव च पूर्वरूपम्। (Ca.Ci.12/10)
- तत्पूर्वरूपं दवथुः सिरायामोऽङ्गगौरवम् । (A.H.Ni.13/30)

Ūṣmā (heat), Davathu (burning sensation), Aṅga Gaurava (heaviness of the body) and Sirāṇāmāyāma (dilatation of vessels) are the premonitory symptoms of Śotha.

Sāmānya Lakṣaṇās

- सगौरवं स्यादनवस्थितत्वं सोत्सेधमुष्माऽथ सिरातनुत्वम्। सलोमहर्षाऽङ्गविवर्णता च सामान्यलिङ्गं श्वयथोः प्रदिष्टम्॥ (Ca.Ci.12/11)

Gaurava (heaviness), Anavasthitatva (instability), Utsedha (protuberance), Ūṣmā (heat), Sirātanutva (thinning of vessels), Lomaharṣā (horripilation) and Aṅgavivarṇa (discoloration of the affected body parts) are the general clinical features of Śvayathu.

Doṣaja bheda Lakṣaṇās of Śotha

Vātaja Śotha Lakṣaṇās

- ⦿ चलस्तनुत्वक्परुषोऽरुणोऽसितः प्रसुप्तिहर्षार्तियुतोऽनिमित्ततः।
  प्रशाम्यति प्रोन्नमति प्रपीडितो दिवाबली च श्वयथुः समीरणात्॥ (Ca.Ci.12/12)
- ⦿ वाताच्छोफश्चलो रूक्षः खररोमाऽरुणासितः॥ सङ्कोचस्पन्दहर्षार्तितोदभेदप्रसुप्तिमान्।
  क्षिप्रोत्थानशमः शीघ्रमुन्नमेत्पीडितस्तनुः॥ स्निग्धोष्णमर्दनैः शाम्येद्रात्रावल्पो दिवा महान्।
  त्वक् च सर्षपलिप्तेव तस्मिंश्चिमिचिमायते॥ (A.H.Ni.13/30-32)
- ⦿ तत्र वातश्वयथुररुणः कृष्णो वा मृदुरनवस्थितास्तोदादयश्चात्र वेदनाविशेषाः। (Su.Ci.23/5)

The clinical features of Vātaja Śvayathu are Cala (unstable or movable), Tanutvakparuṣoaruṇoasita (the affected skin is thin, rough, reddish or blackish in colour), Prasupti (numbness), Harṣā (horripilation), Arti (severe pain); Animittataḥ praśāmyati (subsides without any cause); Pronnamati prapiḍito (depressed on pressure and then elevates immediately), Divābalī (increases or aggravates during day time), Mṛdu (soft) and Anavasthitāstodāya (inconstant pricking pain).

Pittaja Śotha Lakṣaṇās

- ⦿ मृदुः सगन्धोऽसितपीतरागवान् भ्रमज्वरस्वेदतृषामदान्वितः। य उष्यते स्पर्शरुगक्षिरागकृत् स पित्तशोथो भृशदाहपाकवान्॥ (Ca.Ci.12/13)
- ⦿ पीतरक्तासिताभासः पित्तादाताम्ररोमकृत्। शीघ्रानुसारप्रशमो मध्ये प्राग्जायते तनुः॥
  सतृड्दाहज्वरस्वेददवक्लेदमदभ्रमः। शीताभिलाषी विड्भेदी गन्धी स्पर्शासहो मृदुः॥ (A.H.Ni.13/33-34)
- ⦿ पित्तश्वयथुः पीतः सरक्तो वा मृदुः शीघ्रानुसार्यूषादयश्चात्र वेदनाविशेषाः; । (Su.Ci.23/5)

The clinical features of Pittaja Śvayathu are Mṛdu (soft), Sagandha (has odour), Asitapītarāgavān (blackish, yellowish or reddish in colour), Bhrama (giddiness), Jvara (fever), Sveda (perspiration), Tṛṣā (thirst), Madānvita (associated with intoxication), Uṣyate (feeling as if burned), Sparśaruk (pain on touch or tenderness), Akṣirāga (redness of eyes), Bhṛśadāhapākavān (severe burning sensation and suppuration), Śīghrānusārya (spreads quickly) and Ūṣādaya (burning pain).

Kaphaja Śotha Lakṣaṇās

- ⦿ गुरुः स्थिरः पाण्डुररोचकान्वितः प्रसेकनिद्रावमिवह्निमान्द्यकृत्।
  स कृच्छ्रजन्मप्रशमो निपीडितो न चोन्नमेद्रात्रिबली कफात्मकः॥ (Ca.Ci.12/14)

- ⦿ कण्डूमान् पाण्डुरोमत्वक्कठिनः शीतलो गुरुः। स्निग्धः श्लक्ष्णः स्थिरः स्त्यानो निद्राच्छर्द्यग्निसादकृत्॥
  आक्रान्तो नोन्नमेत्कृच्छ्रशमजन्मा निशाबलः। स्रवेन्नासृक् चिरात्पिच्छां कुशशस्त्रादिविक्षतः॥
  स्पर्शोष्णकाङ्क्षी च कफात् ॥ (A.H.Ni.13/35-37)
- ⦿ श्लेष्मश्वयथुः पाण्डुः शुक्लो वा स्निग्धः कठिनः शीतो मन्दानुसारी, कण्ड्वादयश्चात्र वेदनाविशेषाः । (Su.Ci.23/5)

The clinical features of Kaphaja Śvayathu are Guru (heavy), Sthira (stable), Pāṇḍu (pale in colour), Arocakānvita (associated with anorexia), Praseka (salivation), Nidrā (excessive sleep), Vami (vomiting), Vahnimāndyakṛt (subdued digestive fire), Kṛcchrajanma praśamo (appears and disappears slowly), Nipīḍito na ca unnamati (does not elevates after pressure), Rātribalī (increases or aggravates during night time), Śukla (white in colour), Snigdha (slimy), Kaṭhina (hard), Śīta (cold), Mandānusārī (spreads slowly) and Kaṇḍū (itching sensation).

Difference of Doṣaja Śotha

Śotha	Vātaja Śotha	Pittaja Śotha	Kaphaja Śotha
Śotha Guṇa	Cala, Mṛdu, Tanu tvak paruṣo	Mṛdu, Sagandha	Guru, Sthira, Snigdha, Kaṭhina, Śīta,
Śotha Varṇa	Aruṇoasita	Asitapītarāgavān	Pāṇḍu, Śukla
Prasārita	Śīghrānusārī	Śīghrānusārya	Mandānusārī
Śotha lakṣaṇa	Animittataḥ praśāmyati, Pronnamati prapiḍito	Bhṛśadāhapākavān,	Kṛcchrajanma praśamo, Nipīḍito na ca unnamati,
Anubandha lakṣaṇa	Prasupti, Harṣā, Arti	Bhrama, Jvara, Sveda, Tṛṣā, Madānvita, Uṣyate Sparśaruk, Akṣirāga	Arocakānvita, Praseka, Nidrā, Vami, Vahnimāndyakṛt,
Viśeṣa lakṣaṇa	Anavasthitāstodāya	Ūṣādaya	Kaṇḍū
Anupaśaya	Divābalī	Sparśāsaha	Rātribalī

Saṃsargaja and Sannipātaja Śotha Lakṣaṇās

- ⦿ सन्निपातश्वयथुः सर्ववर्णवेदनः; (Su.Ci.23/5)
- ⦿ यथास्वं द्वन्द्वजास्त्रयः। सङ्कराद्धेतुलिङ्गानाम्.. । (A.H.Ni.13/37)

The clinical features of Dvandvaja or Sannipātaja Śvayathu are presence of all the symptoms and pains of respective doṣas appear simultaneously.

Abhighātaja Śotha Lakṣaṇās

- ⦿ अभिघातेन शस्त्रादिच्छेदभेदक्षतादिभिः। हिमानिलोदध्यनिलैर्भल्लातकपिकच्छुजैः॥
  रसैः शूकैश्च संस्पर्शाच्छ्वयथुः स्याद्विसर्पवान्। भृशोष्मा लोहिताभासः प्रायशः पित्तलक्षणः॥ (A.H.Ni.13/38-39)

The causative factors of Abhighātaja Śvayathu are Śastrādicchedabhedakṣatādibhi (cutting, splitting, hitting etc. by sharp weapons etc.), Himānilodadhyanila (by exposing to cold wind and sea wind) and Bhallātakapikacchujaiḥ rasa śukaisca saṃsparśāt (by touching the juice of bhallātaka, hairs of Kapikacchu and sharp spikes) resulting in Visarpavān śvayathu (swelling spreads from place to place), Bhṛśoṣmā (severe burning sensation), Lohitābhāsaḥ (it resembles blood in colour) and Prāyaśaḥ pittalakṣaṇaḥ (mostly has features of Pitta).

Viṣaja Śotha Lakṣaṇās

- विषनिमित्तस्तु गरोपयोगाद्दुष्टतोयसेवनात् प्रकुथितोदकावगाहनात् सविषसत्त्वदिग्ध चूर्णनावचूर्णनाद्वा सविषमूत्रपुरीषशुक्रस्पृष्टानां वा तृणकाष्ठादीनां संस्पर्शनात्, स तु मृदुः क्षिप्रोत्थानोऽवलम्बी चलोऽचलो वा दाहपाकरागप्रायश्च भवति ॥ (Su.Ci.23/5)
- विषजः सविषप्राणिपरिसर्पणमूत्रणात्। दंष्ट्रादन्तनखापातादविषप्राणिनामपि॥
  विण्मूत्रशुक्रोपहतमलवद्वस्त्रसङ्करात्। विषवृक्षानिलस्पर्शाद्गरयोगावचूर्णनात्॥
  मृदुश्चलोऽवलम्बी च शीघ्रो दाहरुजाकरः। (A.H.Ni.13/40-42)

The causative factors of Viṣaja Śvayathu are Garopayogāt (use of slow poison), Duṣṭatoya sevanāt (drinking polluted water), Prakuthitodakāvagāhanāt (immersion or bathing in putrid water), Saviṣasattvadigdhacūrṇenāvacūrṇamādvā (dusting the body with the powders contaminated by any poisonous animals), Saviṣamūtrapurīṣaśukraspṛṣṭānāṃ vā tṛṇakāṣṭhādīnāṃ saṃsparśanāt (by contact with grass and woods contaminated by poisonous urine, faeces or semen) which results in Śvayathu with following features; Mṛdu (soft), Kṣiprotthāno (spreads quickly), Avalambī (hanging), Caloacalo (mobile or fixed) and Dāhapākarāgaprāya (attended with burning sensation, suppuration and redness).

Ekāṅga and Sarvāṅga Śotha

- दोषाः श्वयथुमूर्ध्वं हि कुर्वन्त्यामाशयस्थिताः ॥ पक्वाशयस्था मध्ये तु वर्चःस्थानगतास्त्वधः ।
  कृत्स्नदेहमनुप्राप्ताः कुर्युः सर्वसरं तथा ॥ (Su.Ci.23/6-7)

Doṣas located in Āmāśaya (stomach) give rise to Ūrdhva Śotha (oedema in the upper parts of the body), while doṣas situated in the Pakvāśaya (intestine) result in Madhye sthāna Śotha (oedema in the middle parts of the body). Additionally, doṣas that have reached the Varcaḥsthāna (site of faeces), lead to Adhaḥ śotha (oedema in the lower part of the body). Conversely, doṣas spread throughout the body lead to Sarvasara Śotha (generalized oedema).

Upadrava of Śvayathu

- छर्दिः श्वासोऽरुचिस्तृष्णा ज्वरोऽतीसार एव च। सप्तकोऽयं सदौर्बल्यः शोफोपद्रवसङ्ग्रहः॥ (Ca.Sū.18/18)

Chardi (vomiting), Śvāsa (dyspnoea), Aruci (anorexia), Tṛṣṇā (thirst), Jvara (fever), Atisāra (diarrhea) and Daurbalya (weakness), these seven collectively constitute the complications of Śvayathu.

Sādhyāsādhyata of Śvayathu

- अहीनमांसस्य य एकदोषजो नवो बलस्थस्य सुखः स साधने। (Ca.Ci.12/16)

- नवोऽनुपद्रवः शोफः साध्योऽसाध्यः पुरेरितः॥ (A.H.Ni.13/42)

The patient of Śvayathu having Ahīnamāṃsa (absence of emaciation), Ekadoṣaja (caused by only one doṣa), Nava (recent onset) Anupadrava (absence of complications) and Balastha (afflicting the strong person), then it is Sukhasādhya (easily curable).

- कृशस्य रोगैरबलस्य यो भवेदुपद्रवैर्वा वमिपूर्वकैर्युतः। स हन्ति मर्मानुगतोऽथ राजिमान् परिस्रवेद्धीनबलस्य सर्वगः॥ (Ca.Ci.12/15)

The patient of Śvayathu who becomes Kṛśa (emaciated), Rogairabala (debilitated due to diseases) Bhavetupadravairvā vamipūrvakai (having complications starting with vomiting), Sa hanti Marmānugatoatha rājimān (afflicting vital organs and having streaks over it), Parisravedhīnabalasya (inflicting weak person who has discharge) and Sarvagaḥ (involving whole body), then it leads to death.

- तन्द्रादाहारुचिच्छर्दिमूर्च्छाध्मानाति सारवान्। अनेकोपद्रवयुतः पादाभ्यां प्रसृतो नरम्॥
  नारीं शोफो मुखाद्धन्ति कुक्षिगुह्यादुभावपि। राजीचितः स्रवंश्छर्दि ज्वरश्वासाति सारिणम्॥ (A.H.Śā.5/92-93)

The patient of Śvayathu developing complications such as Tandra (drowsiness), Dāha (burning sensation), Aruci (anorexia), Chardi (vomiting), Mūrcchā (fainting), Ādhmāna (abdominal distension) and Atisāra (diarrhea) etc., Pādābhyāṃ prasṛte naram (starting from the feet in male), Nārīṃ śopho mukhādhanti (starting from the face in female) and Kukṣiguhyādubhāva (starting from abdomen and genitals in both male and female) and spreads all over the body, Rājīcitaḥ (stripes over the swelling), Sravaṃ (discharges) and afflicted with Chardi (vomiting), Jvara (fever), Śvāsa (dyspnoea) and Atisāra (diarrhea) are said to be Asādhya (difficult to cure).

- श्वयथुर्मध्यदेशे यः स कष्टः सर्वगश्च यः ॥ अर्धाङ्गेऽरिष्टभूतश्च यश्चोर्ध्वं परिसर्पति ।
  श्वासः पिपासा दौर्बल्यं ज्वरश्छर्दिररोचकः ॥ हिक्कातीसारकासाश्च शूनं सङ्क्षपयन्ति हि । (Su.Ci.23/7-9)

The patient of Śvayathu who develops swelling in Madhya deśa (middle part of the body) or which is generalized all over the body is Kaṣṭha sādhya (cured with difficulty); and the swelling occurring first at Ardhāṅga (lower half of the body) and extending upwards is incurable; and Śvayathu with complications such as Śvāsa (dyspnoea), Pipāsā (thirst), Daurbalya (weakness), Jvara (fever), Chardi (vomiting), Arocaka (anorexia), Hikkā (hiccup), Atisāra (diarrhea) and Kāsa (cough) leads to death.

In Contemporary science,

Śotha can be correlated to oedema in modern science as it is rather seen as a symptom in many diseases than a disease itself. **Oedema** is swelling of soft tissues due to expansion of interstitial fluid, which contains predominantly water, but protein and cell rich fluid may seen if there is infection or lymphatic obstruction. It may occur in any part of the body including brain, lungs, heart, liver, spleen etc., but most commonly seen in the lower extremities due to gravity.

Oedema may occur in various forms such as localized or generalized oedema. Generalized oedema is commonly caused by cardiac failure, nephrotic syndromes and cirrhosis of liver. Whereas localized oedema is commonly caused by allergic reactions (urticaria or angioedema), infection or inflammation of joints, chronic venous insufficiency, deep vein thrombosis, lymphoedema, may-thurner syndrome (iliac vein compression syndrome), mal-absorption syndrome, obstructive sleep apnea, pregnancy and medications such as anti-hypertensive, antidepressants, chemotherapeutics and NSAIDs.

Diagnosis is based on the clinical evaluation and blood investigations. Clinical history reveal the location, duration; presence of pain, associated symptoms and past medical conditions that may give the clue for diagnosis. Physical examination demonstrates whether the oedema is symmetrical or asymmetrical, pitting or non-pitting and dependant or non-dependant and if associated with warmth, erythema and tenderness. Blood investigations including complete blood count, serum electrolytes, blood urea nitrogen, serum creatinine, liver function tests, serum protein urinalysis helps to rule out the underlying pathology. Other tests like D-dimer, ECG, USG, chest x-ray or CT scan should be done based on the cause.

Short Answer Questions

1. Explain Sāmānya Nidāna and Saṃprāpti of Śotha
2. Explain Nija and Āgantu Śotha Nidāna
3. Differentiate Doṣaja Śotha
4. Describe Viṣaja Śotha
5. Describe Sādhyāsādhyata of Śvayathu

Long Answer Questions

1. Define Śotha, and explain the Nidāna, Saṃprāpti, Pūrvarūpa, Bheda and Lakṣaṇās of Śotha

◆

TOPIC 25

Cardiovascular Disorders

Introduction

Cardiovascular disorders comprise a spectrum of conditions impacting the heart and blood vessels, encompassing coronary artery disease, heart failure, hypertensive heart disease, rheumatic heart disease, cardiomyopathy, arrhythmia, congenital heart disease, valvular heart disease, carditis, aortic aneurysms, peripheral artery disease, thromboembolic disease, and venous thrombosis. This chapter will concentrate on two primary conditions: Coronary Artery Disease (CAD) and Congestive Cardiac Failure (CCF).

CORONARY ARTERY DISEASE (CAD)

Introduction

Coronary artery disease (CAD), also referred to as Coronary heart disease (CHD), Ischemic heart disease (IHD), and Myocardial ischemia, is a condition characterized by insufficient blood and oxygen supply to the myocardium due to occlusion of the coronary arteries. This condition commonly involves the development of arterial plaques that hinder blood flow. It is prevalent in both developed and developing nations. Studies have indicated that CAD accounts for 2.2% of the global disease burden and 32.7% of cardiovascular diseases. In India, CAD poses a significant health challenge, contributing to approximately 25% of the country's mortality rate. The estimated prevalence of CAD in urban and rural areas of India is 7.5% and 4.5%, respectively. The high prevalence of risk factors such as older age, male gender, smoking, hypertension, diabetes mellitus, and hyperlipidemia, as well as family history, has led to an increased incidence of CAD in India.

Pathophysiology:

The primary characteristic of coronary artery disease (CAD) is the formation of atherosclerotic plaque, a buildup of fatty material that narrows the blood vessels and impedes blood flow. This process begins with forming a fatty streak caused by the deposition of lipid-laden macrophages (foam cells) in the subendothelial layer.

Following vascular insult, the intima layer breaks down, facilitating the migration of monocytes to the subendothelial space, where they differentiate into macrophages. These macrophages engulf oxidized low-density lipoprotein (LDL) particles, forming foam cells. The activation of T cells leads to the release of cytokines, which contribute to the pathological process. Furthermore, growth factors released from activated macrophages stimulate the uptake of oxidized LDL particles and collagen by smooth muscle cells, thereby increasing the population of foam cells. This sequence of events culminates in the formation of subendothelial plaque.

Over time, these plaques stabilize, develop fibrous caps, and calcify, potentially leading to angina during increased oxygen demand. Symptoms alleviate during rest as oxygen requirements diminish. A lesion must be at least 70-90% stenosed to cause angina at rest. Rupture of plaques can cause thrombosis, resulting in acute coronary syndrome in the form of unstable angina, NSTEMI, or STEMI, depending on the extent of the insult.

Coronary artery disease (CAD) can be classified into two main categories, namely, Stable Ischemic Heart Disease (SIHD) and Acute Coronary Syndrome (ACS).

Stable Ischemic heart disease (SIHD)

Stable Ischemic Heart Disease (SIHD), also known as Stable coronary artery disease (SCAD) or Chronic coronary artery disease, encompasses various conditions characterized **by a recurring mismatch between myocardial oxygen supply and demand. This typically occurs when prolonged atherosclerotic obstruction in epicardial coronary arteries leads to inadequate blood flow and distal ischemia.** However, atherosclerotic obstruction is not **the sole mechanism;** other pathophysiologic processes can also cause this supply-demand **imbalance, resulting in chronic repetitive ischemia.** These processes include coronary artery **vasospasm, microcirculation dysfunction,** or congenital anomalies..

According to the 2021 American College of Cardiology (ACC) and American Heart Association (AHA) guidelines, Stable Ischemic Heart Disease includes adults with known ischemic heart disease (IHD) experiencing stable pain syndromes (e.g. chronic angina), as well as adults with new-onset, low-risk chest pain (e.g. low-risk unstable angina). Asymptomatic patients diagnosed through non-invasive methods or those whose symptoms are adequately controlled medically or through revascularization.

The typical presentation entails reported **angina pectoris,** commonly referred to as **stable angina,** which is characterized by patients experiencing chest discomfort, often localized on the left side and beneath the sternum. This discomfort typically arises during exertion or emotional stress and is mitigated by rest or the administration of nitroglycerin. The nature of the chest discomfort may vary, encompassing sensations of heaviness, pressure, squeezing, or tightness. Radiation to the left arm, neck, or jaw is frequently noted. Conversely, isolated chest "pain," reproducibility during palpation, variability in breathing, or precise localization by the patient diminishes the probability of ischemia.

Diagnosis of stable ischemic heart disease is based on thorough history and physical examination, along with a baseline ECG to check for signs of ischemia or prior infarction. Stress testing (e.g., treadmill, stress echocardiography, or nuclear stress test) and functional capacity assessment are often necessary, especially in the presence of active symptoms or evidence of ischemia, such as ST changes on ECG. All patients diagnosed with stable ischemic heart disease should undergo an echocardiogram to evaluate ventricular function, as it significantly influences management. This is especially relevant if it reveals reduced left ventricular ejection fraction (LVEF) or regional wall motion abnormalities indicative of ischemia or a previous infarction within a specific coronary artery territory. Routine laboratory tests such as a lipid panel, HbA1c, and renal function should also be routinely performed. While not mandatory for diagnosis, troponin enzyme levels, brain natriuretic peptide (BNP), or N-terminal prohormone of brain natriuretic peptide (NT-proBNP) may offer valuable insights in specific clinical contexts.

Acute coronary syndrome (ACS)

An acute coronary syndrome is characterized by sudden, acute reduction in coronary blood flow, leading to unpredictable patterns of ischemia. Acute Coronary Syndrome (ACS) can be further sub-classified into unstable angina, Non–ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI).

Unstable angina, also referred to as acute coronary insufficiency, preinfarction angina, or intermediate syndrome, is defined by one or more of the following symptoms in patients whose cardiac biomarkers do not meet the criteria for myocardial infarction (MI):

- Prolonged rest angina (usually lasting > 20 minutes)
- Onset of angina of at least class 3 severity in the Canadian Cardiovascular Society (CCS) classification system. The Canadian Cardiovascular Society Classification System for Angina Pectoris is as follows:
  - **Class 1:** Chest pain triggered by strenuous, rapid, or prolonged exertion, not usual physical activities (e.g., walking, climbing stairs)
  - **Class 2:** Chest pain triggered by walking rapidly, walking uphill, climbing stairs rapidly, walking or climbing stairs after meals, cold, wind, or emotional stress
  - **Class 3:** Chest pain triggered by walking 1 or 2 blocks at usual pace and on level ground, or climbing one flight of stairs

◈ **Class 4:** Chest pain triggered by any physical activity, sometimes occurring at rest.

- Increasing angina, such as previously diagnosed angina that has become distinctly more frequent, more severe, longer in duration, or lower in threshold (e.g., increased by ≥ 1 CCS class or to at least CCS class 3).

Diagnosis is based on clinical presentation and further confirmed by ECG changes such as ST-segment depression (in Non-ST-segment elevation MI), ST-segment elevation (in ST-segment elevation MI), or T-wave inversion may occur but they are transient; Cardiac bio markers such as creatine kinase (CK), myoglobin and troponin levels are elevated in unstable angina. Coronary angiography is to evaluate the location and assess the severity of coronary artery lesions.

Myocardial infarction (MI)

Myocardial infarction (MI), also known as a heart attack, occurs when blood flow to the heart is abruptly reduced, causing ischemic necrosis of myocardial cells. Symptoms include chest discomfort or pain that may radiate to other areas, sweating, nausea, vomiting, and fainting. However, some MIs, known as silent MIs, occur without symptoms, particularly in individuals with diabetes mellitus. Acute MI, along with unstable angina, is considered an acute coronary syndrome. Acute MI includes both non–ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI).

Diagnosing MI involves cardiac biomarkers, such as elevated troponin (I or T) and Creatine kinase-MB (CK-MB), which indicate cardiac muscle damage. Electrocardiogram (ECG) changes, including ST-segment elevation or depression, T-wave changes, new Q waves, or a new left bundle branch block, also aid in diagnosis. MIs primarily affect the left ventricle but can extend to the right ventricle or atria.

Infarcts can be classified as transmural (full-thickness) or nontransmural (subendocardial). Transmural infarcts typically show abnormal Q waves on ECG, while nontransmural infarcts exhibit ST-segment and T-wave (ST-T) abnormalities without Q waves. Based on ECG findings, MIs are categorized as ST-segment elevation myocardial infarction (STEMI - ECG showing ST-segment elevation that is not quickly reversed by nitroglycerin) or non-ST-segment elevation myocardial infarction (NSTEMI - ECG changes such as ST-segment depression, T-wave inversion, or both may be present).

CONGESTIVE CARDIAC FAILURE (CCF)

Introduction

Congestive cardiac Failure (CCF), also known as Congestive heart failure (CHF) or Heart failure (HF), is a multifaceted clinical syndrome arising from structural or functional impediments to ventricular blood filling or ejection. It represents a significant global health concern, with an estimated worldwide prevalence of approximately 64 million individuals as of 2023-24. In India, the prevalence of heart failure is considerable, affecting an estimated 1.3 to 4.6 million people. This high prevalence in India can be attributed to a confluence of factors, including a high incidence of ischemic heart disease, hypertension, diabetes, and rheumatic heart disease.

The common causes of congestive heart failure are coronary artery disease, high blood pressure, diabetes mellitus; alcohol abuse and smoking; viral infections and infections like rheumatic fever and typhoid fever, heart valve disorders like aortic and mitral valve diseases, thyroid disorders and congenital lesions like atrial septal defect and ventricular septal defect.

Pathophysiology:

The pathophysiology of Congestive Heart Failure (CHF) involves a complex interplay of mechanisms resulting in impaired ventricular function. CHF arises when the heart's ability to pump blood is compromised, leading to insufficient blood flow to meet the body's metabolic demands. This process initiates with an initial insult or stressor, such as hypertension, coronary artery disease, or cardiomyopathy, which damages the heart muscle or impairs its function, consequently decreasing cardiac output and activating compensatory mechanisms to sustain blood pressure and perfusion.

The sympathetic nervous system is engaged, releasing catecholamines (e.g. adrenaline) that elevate heart rate, contractility, and vascular resistance. The renin-angiotensin-aldosterone system (RAAS) is similarly activated, promoting fluid retention and vasoconstriction. As CHF progresses, these compensatory mechanisms become maladaptive, further contributing to cardiac dysfunction. Increased afterload due to vasoconstriction and decreased compliance due to cardiac remodeling reduces cardiac output. Fluid accumulation and congestion lead to pulmonary oedema, peripheral oedema, and ascites. Decreased cardiac output also triggers renal vasoconstriction, diminishing renal perfusion and exacerbating fluid retention.

The failing heart undergoes remodeling, characterized by hypertrophy, fibrosis, and chamber dilation, which further impairs cardiac function, perpetuating a cycle of decline. Neurohormonal activation, inflammation, and oxidative stress contribute to cardiac damage and dysfunction. The resulting CHF is typified by decreased exercise tolerance, fatigue, dyspnea, and potentially life-threatening complications.

Classification:

Heart failure may present with various symptoms dependent on the underlying type. It is classified based on the heart's pumping capacity, with two primary categories.

- **Heart failure with reduced pumping ability** (heart failure with reduced ejection fraction, also called systolic failure): In this instance, the heart muscle weakens, resulting in inadequate blood circulation and oxygen delivery to the body's organs.

The diminished ejection fraction (EF) may range from mild to severe.

- **Heart failure with preserved pumping ability** (heart failure with preserved ejection fraction, also called diastolic failure): While the heart muscle maintains strength, its tissue stiffens; impeding the heart's filling capacity after contraction. Consequently, insufficient blood flow occurs, particularly during physical activity.
- Furthermore, heart failure can be categorized by the course of the disease.
- **Acute heart failure:** This form can manifest suddenly, often stemming from a severe heart attack or specific heart rhythm irregularities, necessitating urgent medical intervention.
- **Chronic heart failure:** This develops gradually due to underlying medical conditions, such as persistent hypertension.

Additionally, heart failure can be stratified based on the affected side of the heart.

- **Left-sided heart failure** is the most common type and is characterized by the left ventricle's inability to adequately pump blood to the body, resulting in blood accumulation in the vessels emanating from the lungs. Causes may include ischemic heart disease, hypertension, aortic valve disease, mitral incompetence, and cardiomyopathy. Symptoms of left-sided heart failure encompass progressive dyspnoea, particularly during exertion, orthopnoea, paroxysmal nocturnal dyspnoea with or without pulmonary oedema, cardiac asthma, i.e. dyspnoea occurs in the night or early morning hours when the patient is sleeping comfortably and relieved only after sitting for 15-20 minutes. Cheyne-Stokes respiration may be another feature of left heart failure, and other indications such as cyanosis, cold extremities, tachycardia, pulsus alternans and gallop rhythm heard at the lower end of sternum near the apex; pansystolic murmur in the mitral area in mitral incompetence and aortic diastolic or systolic murmur in aortic valve disease. Pulmonary signs include fine crepitations at the bases in early cases and coarse crepitations heard all over the lungs but more at the base in advanced cases. Diagnostic procedures, including X-ray, show cardiomegaly with pulmonary congestion (interstitial oedema and pleural effusion), and the BAT wing appearance of bilateral perihilar shadows suggests the presence of pulmonary oedema. Left ventricular hypertrophy with or without strain is detected by ECG.
- **Right-sided heart failure** is characterized by the insufficient pumping ability of the right ventricle, resulting in inadequate blood flow to the lungs. This condition is attributed to various factors including primary lung diseases (e.g., chronic bronchitis, bronchiectasis, pulmonary tuberculosis, and bronchial asthma), vascular disorders (such as primary pulmonary hypertension), congenital heart diseases (e.g., pulmonary stenosis, atrial septal defect, and ventricular septal defect), valvular heart diseases (including mitral stenosis and tricuspid stenosis), constrictive pericarditis, and as a secondary effect of left heart failure. The characteristic symptoms include generalized weakness, oedema of legs and feet, cough, dyspnoea, anorexia, abdominal distention, pain in the right hypochondrium due to an enlarged liver, oliguria, nocturia, and cyanosis (which appears when right heart failure is secondary to lung diseases like chronic cor pulmonale). Physical examination may reveal clubbing, cyanosis, warm extremities, engorged neck veins, elevated jugular venous pressure, oedema over legs and feet, ascites, and an enlarged tender liver. Severe cases may also exhibit generalized dependent oedema. A pulmonary diastolic murmur may be heard due to pulmonary hypertension, vesicular sounds with prolonged expiration, and bilateral crepitations and rhonchi in the absence of hydrothorax. Diagnostic procedures, including X-ray, show pleural effusion on the right or both sides and an enlarged right ventricle and atrium as seen on an oblique view. ECG may show right axis deviation, tall spiky P waves (P pulmonale), and atrial fibrillation.

- **Biventricular heart failure** occurs as a result of disorders that affect the entire myocardium, such as viral myocarditis, amyloidosis, and Chagas disease, or from long-standing left ventricular failure leading to right ventricular failure. In this condition, both sides of the heart are affected, leading to symptoms similar to those of left-sided and right-sided heart failure, including shortness of breath and fluid buildup.

MCQ's

1. What is another term for Coronary Artery Disease

a) Hypertensive heart disease b) Ischemic heart disease

c) Valvular heart disease d) Congenital heart disease

2. What can the rupture of plaques in CAD lead to

a) Hypertension b) Thrombosis

c) Bradycardia d) Hyperlipidemia

3. What is the initial step in the formation of atherosclerotic plaque

a) Formation of a fibrous cap b) Deposition of lipid-laden macrophages

c) Calcification of the plaque d) Rupture of the intima layer

4. What is ischemia

a) Restriction of blood supply to tissues

b) Overflow of blood to tissues

c) Inadequate deoxygenated blood carrying veins

d) Shortness of breath

5. Which of the following is NOT a sub-classification of Acute Coronary Syndrome (ACS)
   a) Unstable angina
   b) Non–ST-segment elevation myocardial infarction
   c) ST-segment elevation myocardial infarction
   d) Chronic stable angina

6. What is a characteristic symptom of unstable angina
   a) Shortness of breath lasting less than 10 minutes
   b) Prolonged rest angina lasting more than 20 minutes
   c) Chest pain triggered by eating
   d) Headache

7. Which cardiac biomarker is elevated in unstable angina
   a) Creatine kinase (CK) b) Myoglobin
   c) Troponin d) All of the above

8. What is the role of the renin-angiotensin-aldosterone system (RAAS) in CHF
   a) Promoting fluid retention and vasoconstriction
   b) Reducing heart rate
   c) Increasing blood glucose levels
   d) Enhancing oxygen delivery

9. What is a common cause of left-sided heart failure
   a) Chronic bronchitis b) Ischemic heart disease
   c) Pulmonary stenosis d) Tricuspid stenosis

10. Which symptom is NOT typically associated with left-sided heart failure
    a) Progressive dyspnoea b) Orthopnoea
    c) Abdominal distention d) Paroxysmal nocturnal dyspnoea

11. What diagnostic feature on an X-ray suggests the presence of pulmonary oedema in left-sided heart failure
    a) Enlarged right ventricle
    b) Pleural effusion
    c) BAT wing appearance of bilateral perihilar shadows
    d) Right axis deviation

12. What does an ECG typically show in left-sided heart failure
    a) Right axis deviation
    b) Tall spiky P waves
    c) Left ventricular hypertrophy with or without strain
    d) Atrial fibrillation

13. What is a common cause of right-sided heart failure
    a) Aortic valve disease b) Mitral incompetence
    c) Chronic bronchitis d) Cardiomyopathy

14. Which symptom is NOT typically associated with right-sided heart failure
    a) Generalized weakness b) Oedema of legs and feet
    c) Progressive dyspnoea d) Pain in the right hypochondrium

15. What physical examination finding is indicative of right-sided heart failure?
    a) Fine crepitations at the bases b) Cold extremities
    c) Elevated jugular venous pressure d) Gallop rhythm

16. What does an ECG typically show in right-sided heart failure?
    a) Left ventricular hypertrophy b) Right axis deviation
    c) Pansystolic murmur d) Gallop rhythm

Answer Keys

1	b	2	b	3	b	4	a	5	d	6	b	7	d	8	a
9	b	10	c	11	c	12	c	13	c	14	c	15	c	16	b

Short Answer Questions

1. Explain Coronary artery disease
2. Explain acute coronary syndrome
3. Describe stable ischemic heart disease
4. Explain congestive cardiac failure.

TOPIC 26

Kāmalā

Introduction

Kāmalā is one of the Pittaja Nānātmajā Vyādhi and Rakta Pradoṣaja vikāra. Ācārya Caraka considered Kāmalā as an advanced stage of Pāṇḍu roga. Hence he explained both in the same chapter. Ācārya Hārīta explained Kāmalā is one of the eight types of Pāṇḍu roga namely Vātaja, Pittaja, Kaphaja, Sannipātaja, Mṛdbhakṣaṇaja, Kāmalā, Kumbha Kāmalā and Halīmaka. Even though, Ācārya Suśruta considered Kāmalā as a synonym of Pāṇḍu roga, he explained it as an advanced stage of Pāṇḍu roga. Ācārya Vāgbhaṭa stated first that the disease Kāmalā can occur independently.

Nirūkti and Paribhāṣā

Kāmalā is a compound word consisting of the terms Kāma and Lā, which means to grasp the desire.

- ⊙ अत्र कामलेति कामशब्दोऽयं साधारणशब्दविशेषात्स्वल्पे भक्ताद्यभिलाषे प्रवर्तते, तं लातीति कामला। (Hā. Su.U.44/6)

The word Kāmalā generally denotes the term Kāma (desire), but in special sense it diminishes the taste, hunger and appetite for food.

- ⊙ पाण्डुरोगी तु योऽत्यर्थं पित्तलानि निषेवते। तस्य पित्तमसृग्मांसं दग्ध्वा रोगाय कल्पते॥ (Ca.Ci.16/34)

If the patient of Pāṇḍu roga indulges in excessive intake of Pitta aggravating diet and regimen, then the aggravated Pitta burns the Rakta and Māṃsa and produces the disease Kāmalā.

Nidāna and Samprāpti

- ⊙ पाण्डुरोगी तु योऽत्यर्थं पित्तलानि निषेवते। तस्य पित्तमसृग्मांसं दग्ध्वा रोगाय कल्पते॥ (Ca.Ci.16/34)

The Patient of Pāṇḍu roga excessively consumes the Pittalā (Pitta aggravating substances), and then this aggravated Pitta burns the Asṛk (blood) and Māṃsa (muscle tissues) and produces the disease Kāmalā.

- ⊙ यो ह्यामयान्ते सहसाऽन्नमम्लमद्यादपथ्यानि च तस्य पित्तम् ॥ करोति पाण्डुं वदनं विशेषात् पूर्वेरितौ तन्द्रिबलक्षयौ च । (Su.U.44/10-11)

Pitta doṣa aggravates in the one who indulges in Annamamla (food with sour substances), Madya (alcohol) and Apathyā (unwholesome diet) who is not completely cured after suffering from either Pāṇḍurogānte (Pāṇḍu roga) or Anyarogānte (any diseases) and produces Pāṇḍu vadana (paleness of the face) along with the clinical features like Tandrā (drowsiness), Bala kṣaya (decreased strength) as well as the specific symptoms of Pāṇḍu roga.

Samprāpti Ghaṭaka

Doṣa	Pitta Pradhāna Tridoṣa
Dūṣya	Rasa, Rakta, Māṃsa
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhya janya Āma
Srotas	Rasa, Rakta, and Māṃsa vaha
Srotoduṣṭi	Saṅga, Vimārga gamana
Adhiṣṭhāna	Yakṛt and Plīha
Udbhava sthāna	Āmāśaya
Sañcāra sthāna	Raktavaha Yakṛt and Plīha
Vyakta sthāna	Netra, Tvak, Nakha, Mukha, Mūtra, Purīṣa
Rogamārga	Bāhya and Abhyantara

Sāmānya Lakṣaṇās of Kāmalā

- ⊙ हारिद्रनेत्रः स भृशं हारिद्रत्वङ्नखाननः। रक्तपीतशकृन्मूत्रो भेकवर्णो हतेन्द्रियः॥ दाहाविपाकदौर्बल्यसदनारुचिकर्षितः। कामला बहुपित्तैषा कोष्ठशाखाश्रया मता॥ (Ca.Ci.16/35-36)

Hāridra netra tvak nakha ānana (yellowish discoloration of eyes, skin, nails and face), Raktapītasakṛnmūtro (reddish yellow discoloration of faeces and urine), Bhekavarṇa (complexion becomes similar to the colour of a frog), Hatendriya (impaired sense organs), Dāha (burning sensation), Avipāka (indigestion), Daurbalya (weakness), Sadana (weakness) and Aruci (anorexia) are the clinical features of Kāmalā.

Kāmalā Bheda

- कामला बहुपित्तैषा कोष्ठशाखाश्रया मता॥ (Ca.Ci.16/36)

Ācārya Caraka classified Kāmalā in to two types, namely, Koṣṭhāśritā and Śākhāśritā Kāmalā.

Ācārya Suśruta stated that Kumbhakāmalā is a sub type of Kāmalā, and Pānakī, Halīmaka, Lāgharaka and Alasa are the Avasthā Viśeṣa of Kumbhakāmalā. Ācārya Vāgbhaṭa classified Kāmalā into Svatantra and Paratantra Kāmalā.

Alpapittā-Bahupittā Kāmalā

- कोष्ठशाखाश्रया बहुपित्ता पाण्डुरोगपूर्विका भवति, या तु केवलं शाखाश्रया अल्पपित्ता च वक्तव्या सा स्वतन्त्राऽपि भवतीति । (Ca.Ca.Ci.16/34-39)

Cakrapāṇi, while commenting, stated that **Koṣṭhaśākhāśrayā Kāmalā** manifest due to **Bahupittā** (excessive aggravation of Pitta) as the subsequent development of Pāṇḍu roga. In contrast, **Śākhāśrayā Kāmalā** may develop independently with **Alpapittā** (less aggravation of Pitta).

Svatantra-Paratantra Kāmalā

Ācārya Vāgbhaṭa classified Kāmalā into two types namely Svatantra and Paratantra based on the involvement of Pāṇḍu roga. Cakrapāṇi clarifies clearly with the simile that, Kāmalā can occur with the preceding Pāṇḍu Roga or independently without Pāṇḍu Roga, just like Prameha Piḍakās occurs with Prameha or independently without Prameha.

- भवेत्पित्तोल्वणस्यासौ पाण्डुरोगादृतेऽपि च। (A.H.Ni.13/17)

Svatantra (independent) Kāmalā occurs without involvement of Pāṇḍu roga in the person who have aggravation of Pitta. It gets localized into the Raktādayastvak i.e. Śākhāśritā Kāmalā.

- यः पाण्डुरोगी सेवेत पित्तलं तस्य कामलाम्॥ (A.H.Ni.13/15)

Paratantra (dependent) Kāmalā occurs following Pāṇḍu roga and gets localized in the Mahā srotas i.e. Koṣṭhaśākhāśritā Kāmalā.

Koṣṭhaśākhāśritā-Śākhāśritā Kāmalā

Koṣṭhaśākhāśritā Kāmalā

The Nidāna and Lakṣaṇās of Koṣṭhaśākhāśritā Kāmalā are similar to Sāmānya Nidāna and Lakṣaṇās.

Śākhāśritā Kāmalā (Ruddhapatha Kāmalā)

Nidāna and Saṃprāpti:

- रूक्षशीतगुरुस्वादुव्यायामैर्वेगनिग्रहैः॥ कफसम्मूर्च्छितो वायुः स्थानात् पित्तं क्षिपेद्बली।
  हारिद्रनेत्रमूत्रत्वक् श्वेतवर्चास्तदा नरः॥ भवेत् साटोपविष्टम्भो गुरुणा हृदयेन च।
  दौर्बल्याल्पाग्निपार्श्वार्तिहिक्काश्वासारुचिज्वरैः॥ क्रमेणाल्पेऽनुसज्येत पित्ते शाखासमाश्रिते। (Ca.Ci.16/125-128)

Indulgence in Rūkṣa (non-unctuous), Śīta (cold), Guru (heavy), Svādu (sweet) food substances, Vyāyāma (excessive exercise) and Vega nigraha (suppression of natural urges) leads to the aggravation of Vāta associated with Kapha and obstructs the Pitta vaha srotas in Yakṛt and removes out Pitta from its locations and produces the clinical features.

Śākhāśritā Kāmalā Lakṣaṇās:

Hāridra netra tvak mūtra (yellowish discoloration of eyes, skin and urine), Śveta varcas (white coloured faeces) associated with Āṭopa (abdominal distension and gurgling sound), Viṣṭambha (constipation), Guruṇā hṛdayena (heaviness of cardiac region), Daurbalya (weakness), Alpāgni (low digestive fire), Pārśvārti (pain in the flanks), Hikkā (hiccups), Śvāsa (dyspnoea), Aruci (anorexia) and Jvara (fever).

Differentiate Koṣṭhaśākhāśritā Kāmalā and Śākhāśritā Kāmalā

	Koṣṭhaśākhāśritā Kāmalā	Śākhāśritā Kāmalā
Pitta Involvement	Bahupitta Kāmalā	Alpapitta Kāmalā
Śakṛt Varṇa	Rakta Pīta	Tilapiṣṭa sannibhā/ Śveta
Mūtra Varṇa	Rakta Pīta	Hāridra
Netra Tvak Varṇa	Hāridra	Hāridra
Anya Lakṣaṇa	Hatendriya, Dāha, Avipāka, Daurbalya, Sadana and Aruci	Āṭopa, Viṣṭambha, Guruṇā hṛdayena, Daurbalya, Alpāgni, Pārśvārti, Hikkā, Śvāsa, Aruci and Jvara.
Modern Parlance	Pre hepatic or Haemolytic (Non-obstructive) Jaundice	Hepatic and Post hepatic (Obstructive) Jaundice

Kumbhakāmalā

Kumbhakāmalā is named because there will be gross edema of the body along with abdominal swelling (ascites) which resembles a pot in appearance.

- कालान्तरात् खरीभूता कृच्छ्रा स्यात् कुम्भकामला। (Ca.Ci.16/37)
- उपेक्षया च शोफाढ्या सा कृच्छ्रा कुम्भकामला। (A.H.Ni.13/18)

If Kāmalā is not treated properly, in due of course of time, it becomes chronic and getting Kharībhūtā (deep rooted) resulting in excessive dryness of the body or afflict tissue with profound swelling and thus becomes difficult to cure. This condition is called Kumbhakāmalā

- भेदस्तु तस्याः खलु कुम्भसाह्वः शोफो महांस्तत्र च पर्वभेदः ॥ (Su.U.44/11)

Suśruta stated that Kumbhakāmalā is a sub type of Kāmalā with Mahā Śopha (anasarca) and Parvabheda (pain in the joints)

Halīmaka

- ज्वराङ्गमर्दभ्रमसादतन्द्राक्षयान्वितो लाघर(व)कोऽलसाख्यः । तं वातपित्ताद्धरिपीतनीलं हलीमकं नाम वदन्ति तज्ज्ञाः ॥ (Su.U.44/12)

Kumbhakāmalā associated with Jvara (fever), Aṅgamarda (body pain), Bhrama (giddiness), Sāda (malaise), Tandrā (drowsiness) and Kṣaya (emaciation) is known as **Lāgharaka** or **Alasa.**

When the body shows greenish or yellowish or bluish discoloration due to aggravation of Vāta and Pitta, the disease is called **Halīmaka.**

- यदा तु पाण्डोर्वर्णः स्याद्धरितश्यावपीतकः॥ बलोत्साहक्षयस्तन्द्रा मन्द्राग्नित्वं मृदुज्वरः।
  स्त्रीष्वहर्षोऽङ्गमर्दश्च श्वासस्तृष्णाऽरुचिर्भ्रमः॥ हलीमकं तदा तस्य विद्यादनिलपित्ततः। (Ca.Ci.16/132-133)
- हरितश्यावपीतत्वं पाण्डुरोगे यदा भवेत्। वातपित्ताद्भ्रमस्तृष्णा स्त्रीष्वहर्षो मृदुर्ज्वरः।
  तन्द्राबलानलभ्रंशो लोढरं तं हलीमकम्॥ अलसं चेति शंसन्ति .... ॥ (A.H.Ni.13/18-19)

When in the body of Pāṇḍu Rogi greenish or bluish or yellowish discoloration appears along with Bala utsāha kṣaya (loss of strength and enthusiasm), Bhrama (giddiness), Tṛṣṇā (thirst), Strīṣvaharṣā (lack of desire in women), Aṅgamarda (body ache), Śvāsa (dyspnoea), Aruci (anorexia), Mṛdu Jvara (mild fever), Tandrā (drowsiness) and Mandāgni or anala bhramśa (weakness of the digestive fire) due to aggravation of Vāta and Pitta, the disease is called **Loḍhara** or **Halīmaka** or **Alasa.**

Pānakī

- सन्तापो भिन्नवर्चस्त्वं बहिरन्तश्च पीतता। पाण्डुता नेत्ररोगश्च पानकीलक्षणं वदेत् ॥ (Ḍa.Su.U.44/12)

Pānakī is the Avasthā bheda of Kumbhakāmalā and the clinical features of Pānakī are Santāpa (increased body temperature), Bhinna varcas (diarrhoea), Bahiranta pītatā (yellowishness of the body both internally and externally), Pāṇḍutā (paleness) and Netra roga (disorders of eyes)

Upadrava of Kāmalā

- उपद्रवास्तेष्वरुचिः पिपासा छर्दिर्ज्वरो मूर्धरुजाऽग्निसादः । शोफस्तथा कण्ठगतोऽबलत्वं मूर्च्छा क्लमो हृद्यवपीडनं च ॥ (Su.U.44/13)

Aruci (anorexia), Pipāsā (thirst), Chardi (vomiting), Jvara (fever), Mūrdharujā (headache), Agnisāda (indigestion), Kanthagato Śopha (oedema in the throat region), Abalatvaṃ (loss of strength), Mūrcchā (fainting), Klama (fatigue) and Hṛdaya pīḍa (pain in the heart region) are the complication of Kāmalā.

Sādhyāsādhyata of Kāmalā

- कृष्णपीतशकृन्मूत्रो भृशं शूनश्च मानवः॥ सरक्ताक्षिमुखच्छर्दिविण्मूत्रो यश्च ताम्यति।
  दाहारुचितृषानाहतन्द्रामोहसमन्वितः॥ नष्टाग्निसञ्ज्ञः क्षिप्रं हि कामलावान् विपद्यते। (Ca.Ci.16/37-39)

Kṛṣṇapīta sakṛnmūtro (blackish or yellowish discoloration of the faeces and urine); Bhṛśam śūna (severe oedema), Saraktākṣimukhacchardiviṇmūtro (eyes, face, vomit, faeces and urine becomes reddish like blood or mixed with blood), Tāmyati (feels darkness before eyes), Dāha (burning sensation), Aruci (anorexia), Tṛṣṇā (morbid thirst), Ānāha (abdominal distension with constipation), Tandrā (drowsiness) and Moha (delusion) and Naṣṭāgnisamjña (loss of digestive fire and consciousness) are the Asādhya lakṣaṇās of Kāmalā and it leads to death.

In Contemporary science,

Kāmalā can be correlated with Jaundice. (Please refer Topic 28)

Short Answer Questions

1. Explain Sāmānya Nidāna and Saṃprāpti of Kāmalā
2. Explain Koṣṭhaśākhāśritā Kāmalā
3. Explain Śākhāśritā Kāmalā
4. Differentiate Koṣṭhaśākhāśritā and Śākhāśritā Kāmalā
5. Describe Svatantra- Paratantra Kāmalā and Alpapittā-Bahupittā Kāmalā

Long Answer Questions

1. Define Kāmalā, and explain the Nidāna, Saṃprāpti, Pūrvarūpa and Lakṣaṇās of Kāmalā

◆

TOPIC 27

Udara

Introduction

Udara is one of the diseases of Udakavaha srotas and occurs as a result of Mandāgni and Malavṛddhi. Udara roga has been named on the basis of its Sthāna (pathological site) and it is regarded as one among the Aṣṭamahāgada. Ācārya Caraka explained Udara after Śotha because Udara is considered as a type of Śotha (Śotha prabheda). Vāgbhaṭa explained Udara after Ānāha because Udara is one of the symptoms of Ānāha.

Nirūkti and Paribhāṣa

- अत्रोदरशब्देनोदरस्थो रोग उच्यते । (SKD)
- उदरस्थत्वाद्वास्योदरशब्दवाच्यता । (Vācaspatyam)
- अत्रोदरस्थो रोगोऽप्युदरशब्देन वाच्यस्तद्गतत्वात्॥ (Āḍ.Śā.P.7/51)

The disease which manifests in Udara is termed as Udara roga.

- उदररोगः उदरी इति ख्याते रोगे । (SKD)

The disease which produces large abdomen is called as Udara roga.

- पूरणलक्षणमुदरमिति। (Ḍa.Su.Sū.21/34)

That which does Pūraṇa (filling) of abdomen is called as Udara roga.

- उदाराण्युत्सेधसामान्याच्छोथप्रभेदा एव॥ (Ca.Ca.Sū.19/3)
- उदरोत्सेधसाधर्म्यादुदर । (Ma.M.N.35/1)

The disease which produces Udarotsedha (swelling of abdomen) is called as Udara roga and it is considered as a type of Śotha.

Sāmānya Nidāna and Saṃprāpti of Udara

- अत्युष्णलवणक्षारविदाह्यम्लगराशनात्। मिथ्यासंसर्जनाद्रूक्षविरुद्धाशुचिभोजनात्॥
  प्लीहार्शोग्रहणीदोषकर्शनात् कर्मविभ्रमात्। क्लिष्टानामप्रतीकाराद्रौक्ष्याद्वेगविधारणात्॥
  स्रोतसां दूषणादामात् सङ्क्षोभादतिपूरणात्। अर्शोबालशकृद्रोधादन्त्रस्फुटनभेदनात्॥
  अतिसञ्चितदोषाणां पापं कर्म च कुर्वताम्। उदराण्युपजायन्ते मन्दाग्नीनां विशेषतः॥ (Ca.Ci.13/12-15)

Atyuṣṇalavaṇakṣāravidāhyamlagarāśanāt (excessive intake of food substances which are hot, saline, alkaline, causing burning sensation during digestion, sour and poisoned food), Mithyāsaṃsarjanāt (improper dietetic regimen after purificatory therapy), Rukṣaviruddhāśuci bhojanāt (intake of non-unctuous, contrary and unhygienic food substances), Karśanāt (emaciation) due to Plīha, Arśas and Grahaṇī doṣa, Karmavibhramāt (improper application of purificatory therapy), Kliṣṭānāmapratikārāt (avoidance of treatment of persistent disorders), Raukṣyāt (roughness), Vegavidhāraṇāt (suppression of natural urges), Srotasāṃ dūṣaṇāt (morbidity of channels), Āmāt (Āmadoṣa), Saṅkṣobhāt (agitation), Atipūraṇāt (over filling or satiation), Arśobālaśakṛdrodhāt (obstruction to the passage by piles, hairs and faeces), Antrasphuṭana bhedanāt (perforation or rupture of intestines), Atisañcitadoṣāṇāṃ (excessive accumulation of morbidity), Pāpakarma (sinful acts) and especially Mandāgni (subdued digestive fire) are the causative factors of the disease Udararoga.

- रुद्ध्वा स्वेदाम्बुवाहीनि दोषाः स्रोतांसि सञ्चिताः। प्राणाग्न्यपानान् सन्दूष्य जनयन्त्युदरं नृणाम्॥ (Ca.Ci.13/20)

Due to the above said causative factors, the accumulated doṣas in the srotas obstructs the Sveda and Ambuvaha srotas and afflicts the Prāṇa, Agni and Apāna Vāta and produces the disease Udara.

- रोगाः सर्वेऽपि मन्देऽग्नौ सुतरामुदराणि तु। अजीर्णान्मलिनैश्चान्नैर्जायन्ते मलसञ्चयात्॥
  ऊर्ध्वाधो धातवो रुद्ध्वा वाहिनीरम्बुवाहिनीः। प्राणाग्न्यपानान् सन्दूष्य कुर्यस्त्वङ्मांससन्धिगाः॥
  आध्माप्य कुक्षिमुदरम् .....॥ (A.H.Ni.12/1-3)

Generally, Mandāgni (subdued digestive fire) is the cause for all the diseases especially Udara roga because it occurs due to Ajīrṇā (indigestion), Malinānna (contaminated food) and Malasañcayāt (accumulation of faeces). By these factors, the aggravated doṣas obstructs the Ūrdhva (upward) and Adhaḥ (downward) passage of Ambuvaha srotas and it vitiates the Prāṇa, Agni and Apāna Vāta and gets lodged in between the Tvak and Māṃsa and produces Kukṣī ādhmāna (distension of abdomen) and resulting in Udara roga.

Saṃprāpti Ghaṭaka of Udara

Doṣa	Tridoṣa
Dūṣya	Rasa
Agni	Jaṭharāgni Dhātvāgni māndhya

Āma	Agni māndhya janya Āma
Srotas	Anna, Purīṣa, Sveda, and Ambu vaha
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Sirāḥ
Udbhava sthāna	Āmāśaya
Vyakta sthāna	Udara
Adhiṣṭhāna	Udara (Tvakmāṃsāntara)
Rogamārga	Abhyantara

Pūrvarūpa of Udara

- क्षुन्नाशः स्वाद्वतिस्निग्धगुर्वन्नं पच्यते चिरात्। भुक्तं विदह्यते सर्वं जीर्णाजीर्णं न वेत्ति च॥
  सहते नातिसौहित्यमीषच्छोफश्च पादयोः। शश्वद्बलक्षयोऽल्पेऽपि व्यायामे श्वासमृच्छति॥
  वृद्धिः पुरीषनिचयो रूक्षोदावर्तहेतुका। बस्तिसन्धौ रुगाध्मानं वर्धते पाट्यतेऽपि च॥
  आतन्यते च जठरमपि लघ्वल्पभोजनात्। राजीजन्म वलीनाश इति लिङ्गं भविष्यताम्॥ (Ca.Ci.13/16-19)

Kṣunnāśa (loss of appetite), Svādvatisnigdhagurvannaṃ pacyate cirāt (delayed digestion of sweet, excessive unctuous and heavy foods), Bhuktaṃ vidahyate sarvaṃ (digested food producing burning sensation), Jīrṇājīrṇaṃ na vetti (inability to understand the digestion and indigestion of the food), Sahate nātisauhityaṃ (intolerance to over-eating), Īṣaccophaśca pādayo (slight swelling in the legs), Śaśvadbalakṣaya (constant reduction of strength), Alpeapi vyāyāme śvāsamṛcchatti (dyspnoea even in mild exertion), Vṛddhiḥ purīṣanicayo (abdominal distension due to accumulation of faeces) Rūkṣodāvartahetukā (upward movement of Vāta due to non-unctuousness), Bastisandhau ruja (pain in pelvic joints), Ādhmāna vardhate pāṭyateapi (bursting type of abdominal pain over the distension of the abdomen), Ātanyate ca jaṭharamapi laghvalpabhojanāt (distension of the abdomen even with little quantity of food), Rajījanma (appearance of network of veins) and Valīnāśa (disappearance of abdominal folds) are the premonitory symptoms of the disease Udara.

- तत्पूर्वरूपं बलवर्णकाङ्क्षावलीविनाशो जठरे हि राज्यः ॥ जीर्णापरिज्ञानविदाहवत्यो बस्तौ रुजः पादगतश्च शोफः । (Su.Ni.7/7-8)

Balavarṇakāñkṣā vināśa (loss of strength, complexion and appetite), Valīvināśa (disappearance of abdominal folds), Jaṭhare hi rājyaḥ (appearance of network of veins over the abdomen), Jīrṇāparijñāna (inability to understand the digestion process), Vidāhavatyo (digested food producing burning sensation), Basti rujaḥ (pain in the bladder) and Pādagata śopha (swelling of the legs) are the premonitory symptoms of the disease Udara.

Sāmānya Lakṣaṇās of Udara

- कुक्षेराध्मानमाटोपः शोफः पादकरस्य च। मन्दोऽग्निः श्लक्ष्णगण्डत्वं कार्श्यं चोदरलक्षणम्॥ (Ca.Ci.13/21)
- तेनार्ताः शुष्कताल्वोष्ठाः शूनपादकरोदराः। नष्टचेष्टाबलाहाराः कृशाः प्रध्मातकुक्षयः॥
  स्युः प्रेतरूपाः पुरुषाः.....॥ (A.H.Ni.12/4-5)

Kukṣerādhmāna / Pradhmātakukṣayaḥ (distension of abdomen), Ātopa (gurgling sound in the abdomen), Pādakara śopha / Śūnapādakarodara (swelling of hands, legs and abdomen), Mandāgni (subdued digestive fire), Ślakṣṇagaṇḍatvaṃ (smoothness of cheeks), Kārśya / Kṛśā (emaciation), Śuṣkatālvoṣṭha (dryness of lips and palate), Naṣṭaceṣṭābalāhārā (loss of activities, strength and desire for food) and Preta rūpa puruṣā (appears like a cadaver) are the general clinical features of Udara roga.

- आध्मानं गमनेऽशक्तिर्दौर्बल्यं दुर्बलाग्निता । शोफः सदनमङ्गानां सङ्गो वातपुरीषयोः ।
  दाहस्तृष्णा च सर्वेषु जठरेषु भवन्ति हि ॥ (Su.Ni.7/24)

Ādhmāna (distension of abdomen), Gamaneaśakti (inability to move), Daurbalya (weakness), Durbalāgni (weak digestive fire), Śopha (swelling), Aṅgasadana (lassitude in the limbs), Saṅgavātapurīṣayo (obstruction to the flatus and faeces), Dāha (burning sensation), Tṛṣṇā (thirst) are the general features appearing in all types of Udara.

Udara bheda

- पृथग्दोषैः समस्तैश्च प्लीहबद्धक्षतोदकैः। सम्भवन्त्युदराण्यष्टौ.....। (Ca.Ci.13/22)
- पृथक् समस्तैरपि चेह दोषैः प्लीहोदरं बद्धगुदं तथैव । आगन्तुकं सप्तममष्टमं च दकोदरं चेति वदन्ति तानि ॥ (Su.Ni.7/4)
- ....अष्टधा तच्च भिद्यते। पृथग्दोषैः समस्तैश्च प्लीहबद्धक्षतोदकैः॥ (A.H.Ni.12/3)

Udara roga is classified in to eight types namely one from each doṣa i.e. Vātaja, Pittaja and Kaphaja; Sannipātaja, Plīhodara, Baddhodara, Kṣatodara and Udakodara. Suśruta mentioned Āgantuka and Dakodara instead of Kṣatodara and Udakodara.

Viśeṣa Nidāna and Lakṣaṇās of Udara

Vātodara Nidāna and Lakṣaṇās

- रूक्षाल्पभोजनायासवेगोदावर्तकर्शनैः। वायुः प्रकुपितः कुक्षिहृद्बस्तिगुदमार्गगः॥
  हत्वाऽग्निं कफमुद्धूय तेन रूद्धगतिस्ततः। आचिनोत्युदरं जन्तोस्त्वङ्मांसान्तरमाश्रितः। (Ca.Ci.13/23-24)

Rūkṣālpabhojana (intake of non-unctuous and less quantity of food), Āyāsa (exertion), Vega (suppression of natural urges), Udāvarta (upward movement of Vāta) and Karśanaiḥ (involving in emaciating factors such as fasting etc.,) leads to aggravation of Vāta and it reaches the Kukṣi (abdomen), Hṛdaya (chest), Basti (pelvis) and Gudamārga (anal passage) and it further destroys the Agni (digestive fire) and aggravates Kapha which inturn gets obstructed in its passage and enters the space between the Tvak (skin) and Māṃsa (muscles) and produces the disease Vātodara.

- कुक्षिपाणिपादवृषणश्वयथुः, उदरविपाटनम्, अनियतौ च वृद्धिह्रासौ, कुक्षिपार्श्वशूलोदावर्ताङ्गमर्दपर्वभेदशुष्ककास कार्श्यदौर्बल्यारोचकाविपाकाः, अधोगुरुत्वं, वातवर्चोमूत्रसङ्गः, श्यावारुणत्वं च नखनयनवदनत्वङ्मूत्रवर्चसाम्, अपि चोदरं तन्वसितराजीसिरासन्ततम्, आहतमाध्मातदृतिशब्दवद्भवति, वायुश्चोर्ध्वमधस्तिर्यक् च सशूलशब्दश्चरति, एतद्वातोदरमिति विद्यात्। (Ca.Ci.13/25)

- तत्र वातोदरे शोफः पाणिपान्मुष्ककुक्षिषु॥ कुक्षिपार्श्वोदरकटीपृष्ठरुक् पर्वभेदनम्।
  शुष्ककासोऽङ्गमर्दोऽधोगुरुता मलसङ्ग्रहः॥ श्यावारुणत्वगादित्वमकस्माद्वृद्धिह्रासवत्।
  सतोदभेदमुदरं तनुकृष्णसिराततम्॥ आध्मातदृतिवच्छब्दमाहतं प्रकरोति च।
  वायुश्चात्र सरुक्शब्दो विचरेत्सर्वतोगतिः। (A.H.Ni.12/12-15)

The clinical features of Vātodara are Kukṣipāṇipādavṛṣaṇa śvayathu (swelling of abdomen, hands, legs and scrotum), Udaravipāṭana (tearing type of pain in the abdomen), Aniyatau ca vṛddhihrāsau (irregular increased and decrease of abdomen), Kukṣipārśvaśūla (pain in the abdomen and flanks), Udāvarta (upward movement of Vāta), Aṅgamarda (body ache), Parvabheda (pain in the small joints), Śuṣkakāsa (dry cough), Kārśya (emaciation), Daurbalya (debility), Arocaka (anorexia), Avipāka (indigestion), Adhogurutvaṃ (heaviness in the lower body parts), Vātavarcomūtrasaṅga (obstruction of flatus, faeces and urine), Śyāvāruṇatvaṃ ca nakhanayanavadanatvakmūtravarcasām (dark brown or reddish discoloration of the nails, eyes, mouth, skin, urine and faeces), Tanvasitarājīsirāsantatam (appearance of thin and dark coloured network of veins over the abdomen), Āhatamādhmātadṛtiśabdavat (on strike produces sound like that of air filled leather bag) and Vāyuścordhvamadhastriyak ca saśūlaśabdaścarati (Vāta moves in upward, downward and oblique direction associated with pain and sound).

- सङ्गृह्य पार्श्वोदरपृष्ठनाभीर्यद्वर्धते कृष्णसिरावनद्धम् ॥ सशूलमानाहवदुग्रशब्दं सतोदभेदं पवनात्मकं तत् । (Su.Ni.7/8-9)

In Vātaja Udara, the aggravated Vāta gets located in the Pārśva (flanks), Udara (abdomen), Pṛṣṭha (back) and Nābhī (umbilical region) and results in appearance of Kṛṣṇa sirāvanaddham (blackish vein) associated with Śūla (pain), Ānāha (suppression of the stool and urine), Ugraśabda (loud noise), Toda (pricking pain) and Bheda (cutting pain)

Pittodara Nidāna and Lakṣaṇās

- कट्वम्ललवणात्युष्णतीक्ष्णाग्न्यातपसेवनैः। विदाह्यध्यशनाजीर्णैश्चाशु पित्तं समाचितम्॥
  प्राप्यानिलकफौ रुद्ध्वा मार्गमुन्मार्गमास्थितम्। निहन्त्यामाशये वह्निं जनयत्युदरं ततः॥ (Ca.Ci.13/26-27)

Kaṭvamlalavaṇātyuṣṇatīkṣṅa (excessive intake of pungent, sour, saline, very hot and sharp substances), Agnyātapasevana (exposure to fire and sun heat), Vidāha (use of food substances causing burning sensation during digestion), Adhyaśana (intake of food prior to digestion of earlier taken food) and Ajīrṇa (indigestion) leads to aggravation of Pitta and it reaches the

Vāta and Kapha sthāna and obstructs their passage and moves upwards and gets inhabitated there and impairs the Agni (digestive fire) and produces the disease Pittodara.

- दाहज्वरतृष्णामूर्च्छातीसारभ्रमाः, कटुकास्यत्वं, हरितहारिद्रत्वं च नखनयनवदनत्वङ्मूत्रवर्चसाम्, अपि चोदरं नीलपीतहारिद्रहरितताम्रराजीसिरावनद्धं, दह्यते, दूयते, धूप्यते, ऊष्मायते, स्विद्यते, क्लिद्यते, मृदुस्पर्शं क्षिप्रपाकं च भवति; एतत् पित्तोदरमिति विद्यात्॥ (Ca.Ci.13/28)

- पित्तोदरे ज्वरो मूर्च्छा दाहस्तृट् कटुकास्यता। भ्रमोऽतिसारः पीतत्वं त्वगादावुदरं हरित्॥
  पीतताम्रसिरानद्धं सस्वेदं सोश्म दह्यते। धूमायति मृदुस्पर्शं क्षिप्रपाकं प्रदूयते॥ (A.H.Ni.12/16-17)

The clinical features of Pittodara are Dāha (burning sensation), Jvara (fever), Tṛṣṇā (thirst), Mūrcchā (fainting), Atīsāra (diarrhoea), Bhramā (giddiness), Kaṭukāsyatva (pungent taste in the mouth) Haritahāridratvaṃ ca nakhanayanavadanatvakmūtravarcasām (greenish or yellowish discoloration of the nails, eyes, mouth, skin, urine and faeces), Nīlapītahāridraharitatāmra rājīsirāvanaddhaṃ (appearance of bluish, yellowish, deep yellow, greenish or coppery coloured network of veins over the abdomen), Dahyate (burning sensation), Dūyate (pain), Dhūpyate (as if smoke is coming out from mouth), Uṣmāyate (feels as if getting heated by the fire), Svidyate (gets drenched with perspiration), Klidyate (damp or moistens), Mṛdusparśa (abdomen is soft to touch) and Kṣiprapāka (gets suppurated quickly).

- यच्चोषतृष्णाज्वरदाहयुक्तं पीतं सिरा भान्ति च यत्र पीताः ॥ पीताक्षिविण्मूत्रनखाननस्य पित्तोदरं तत्त्वचिराभिवृद्धि । (Su.Ni.7/9-10)

The clinical features of Pittaja Udara are Coṣa (sucking type of pain), Tṛṣṇā (thirst), Jvara (fever), Dāha (burning sensation), Pīta (yellowish discoloration of swollen skin of the abdomen), Sirābhānti cayatra Pītāh (appearance of yellowish veins), Pītākṣiviṇmūtranakhānana (yellow colour of the eyes, faeces, urine, nails and face) and Tattva Cirābhivṛddhi (increases rapidly).

Kaphodara Nidāna and Lakṣaṇās

- अव्यायामदिवास्वप्नस्वाद्वतिस्निग्धपिच्छिलैः। दधिदुग्धौदकानूपमांसैश्चाप्यतिसेवितैः॥
  क्रुद्धेन श्लेष्मणा स्रोतःस्वावृतेष्वावृतोऽनिलः। तमेव पीडयन् कुर्यादुदरं बहिरन्त्रगः ॥ (Ca.Ci.13/29-30)

Avyāyāma (lack of exercise), Divāsvapna (sleeping during day time), Svādvatisnigdhapicchilaiḥ (excessive intake of sweet, unctuous and slimy food substances), Dadhi (use of curd), Dugdha (use of milk) and Audakānūpamāṃsaiścāpyaatisevitaiḥ (excessive intake of meat of aquatic and marshy animals) leads to aggravation of Kapha and it obstructs the Srotas and passage of Vāta. Thus the obstructed Vāta expels the Kapha out of the intestines and produces the disease Kaphodara.

- गौरवारोचकाविपाकाङ्गमर्दाः, सुप्तिः, पाणिपादमुष्कोरुशोफः, उत्क्लेशनिद्राकासश्वासाः, शुक्लत्वं च नखनयनवदनत्वङ्मूत्रवर्चसाम्; अपि चोदरं शुक्लराजीसिरासन्ततं, गुरु, स्तिमितं, स्थिरं, कठिनं च भवति; एतच्छलेष्मोदरमिति विद्यात्॥ (Ca.Ci.13/31)

- श्लेष्मोदरेऽङ्गसदनं स्वापः श्वयथुगौरवम्। निद्रोत्क्लेशारुचिश्वासकासशुक्लत्वगादिता॥

उदरं स्तिमितं श्लक्ष्णं शुक्लराजीततं महत्। चिराभिवृद्धि कठिनं शीतस्पर्शं गुरु स्थिरम्॥ (A.H.Ni.12/18-19)

The clinical features of Kaphodara are Gaurava (heaviness of the body), Arocaka (anorexia), Avipāka (indigestion), Aṅgamarda (body ache), Supti (numbness), Pāṇipādamuṣkoruśopha (swelling of hands, legs, scrotum and thighs), Utkleśa (nausea) Nidrā (over sleep), Kāsa (cough), Śvāsa (dyspnoea), Śuklatvaṃ ca nakhanayanavadana tvakmūtravarcasām (whitish discoloration of the nails, eyes, mouth, skin, urine and faeces), Śuklarājīsirāsantatam (appearance of whitish coloured network of veins over the abdomen), Guru (heaviness of the abdomen), Stimitaṃ (feeling as if abdomen is covered with wet cloth), Sthiraṃ (immobility of the abdomen) and Kaṭhinaṃ (hardness of the abdomen).

- यच्छीतलं शुक्लसिरावनद्धं गुरु स्थिरं शुक्लनखाननस्य ॥ स्निग्धं महच्छोफयुतं ससादं कफोदरं तत्तु चिराभिवृद्धि । (Su.Ni.7/10-11)

The clinical features of Kaphaja Udara are Śītalaṃ (feeling of coldness), Śuklasirāvanaddhaṃ (appearance of white veins), Guru (feeling of heaviness), Sthira (hard), Śuklanakhānana (white colour of nails and face), Snigdhaṃ (sliminess), Mahaccopha (marked swelling), Sasādaṃ (lassitude) and Tattu Cirābhivṛddhi (increases slowly).

Differentiate Doṣaja Udara

Udara	Vātaja Udara	Pittaja Udara	Kaphaja Udara
Vedanā	Udaravipāṭana	Dāha	Śītala, Gaurava
Varṇa	Śyāva aruṇatvaṃ of Nakha, Nayana, Vadana, Tvak, Mūtra, Varcas	Haritahāridratvaṃ of Nakha, Nayana, Vadana, Tvak, Mūtra, Varcas	Śuklatvaṃ of Nakha, Nayana, Vadana, Tvak, Mūtra, Varcas
Śvayathu Lakṣaṇa	Kukṣipāṇipādavṛṣaṇa śvayathu	-	Pāṇipādamuṣkoruśopha
Udara Lakṣaṇa	Aniyatau ca vṛddhihrāsau	Mṛdusparśa, Tattva Cirābhivṛddhi	Kaṭhinaṃ, Tattu Cirābhivṛddhi
Anubandha Lakṣaṇa	Kukṣipārśvaśūla, Udāvarta, Aṅgamarda, Parvabheda, Śuṣkakāsa, Kārśya, Daurbalya, Arocaka, Avipāka, Adhogurutvaṃ, Vātavarcomūtrasaṅga,	Jvara, Tṛṣṇā, Mūrcchā, Atīsāra, Bhramā, Kaṭukāsyatva	Arocaka, Avipāka, Aṅgamarda, Supti, Utkleśa, Nidrā, Kāsa, Śvāsa
Sirā Lakṣaṇa	Tanvasita rājīsirāsantatam	Nīla pīta hāridra harita tāmra rājīsirāvanaddhaṃ	Śuklarājīsirāsantatam
Viśeṣa Lakṣaṇa	Āhatamādhmātadṛtiśabdavat, Vāyuścordhvamadhastiyak ca saśūlaśabdaścarati	Dahyate, Dūyate, Dhūpyate, Uṣmāyate, Svidyate, Klidyate, Kṣiprapāka	Guru, Stimitaṃ, Sthiraṃ, Mahacchopha

Sannipātodara Nidāna and Lakṣaṇās

- दुर्बलाग्नेरपथ्यामविरोधिगुरुभोजनैः। स्त्रीदत्तैश्च रजोरोमविण्मूत्रास्थिनखादिभिः॥
  विषैश्च मन्दैर्वाताद्याः कुपिताः सञ्चयं त्रयः। शनैः कोष्ठे प्रकुर्वन्तो जनयन्त्युदरं नृणाम्॥ (Ca.Ci.13/32-33)
- त्रिदोषकोपनैस्तैस्तैः स्त्रीदत्तैश्च रजोमलैः। गरदूषीविषाद्यैश्च सरक्ताः सञ्चिता मलाः॥
  कोष्ठं प्राप्य विकुर्वाणाः शोषमूर्च्छाभ्रमान्वितम्। कुर्युस्त्रिलिङ्गमुदरं शीघ्रपाकं सुदारुणम्॥
  बाधते तच्च सुतरां शीतवाताभ्रदर्शने। (A.H.Ni.12/20-22)

The person with Durbalāgni (impaired digestive fire) indulging in Apathya (unwholesome), Āma (uncooked), Virodhi (contrary), Guru bhojanaiḥ (heavy food substances); Strīdattaiśca rajoromaviṇmūtrāsthinakhādhibhi (ignorant women offer food mixed with menstrual blood, hair, feces, urine, bones, and nails etc.); Viṣaiśca mandai (slow poisoning); leads to vitiation of Vātādi doṣas which gets accumulated in the Koṣṭha gradually producing the disease Sannipātodara.

- सर्वेषामेव दोषाणां समस्तानि लिङ्गान्युपलभ्यन्ते, वर्णाश्च सर्वे नखादिषु, उदरमपि नानावर्णराजीसिरासन्ततं भवति; एतत् सन्निपातोदरमिति विद्यात्॥ (Ca.Ci.13/34)

Sarveśāmeva doṣāṇāṃ samasthāni liṅgānyupalabhyante i.e. manifestation of all the clinical features of all the three doṣas, Varṇāśca sarve nakhādiśu (appearance of all type of colours in the nails, etc.) and Nānavarṇa rājīsirāsantataṃ (appearance of various colour network of veins over the abdomen) are the clinical features of Sannipātodara.

- स्त्रियोऽन्नपानं नखरोममूत्रविडार्तवैर्युक्तमसाधुवृत्ताः ॥ यस्मै प्रयच्छन्त्यरयो गरांश्च दुष्टाम्बुदूषीविषसेवनाद्वा । तेनाशु रक्तं कुपिताश्च दोषाः कुर्वन्ति घोरं जठरं त्रिलिङ्गम् ॥ तच्छीतवाताभ्रसमुद्भवेषु विशेषतः कुप्यति दह्यते च स चातुरो मूर्च्छति सम्प्रसक्तं पाण्डुः कृशः शुष्यति तृष्णया च ॥ प्रकीर्तितं दूष्युदरं तु घोरं ... । (Su.Ni.7/11-14)

Striyoannapānaṃ Nakharomamūtraviḍārtava (women with bad behavior offer food and drinks mixed with nails, hair, urine, feces, menstrual blood), Prayacchantyarayo Garā (in whom enemies give Gara poison), Duṣtāmbu duṣīviṣa sevanādvā (intake of contaminated water or slow poison) aggravates the Rakta and three doṣas and produces the dreadful disease Sannipātodara and which aggravates during Śītavātābhrasamudbhaveṣu (cold, windy and cloudy days) and produces the following features such as Dahyate (burning pain in the abdomen), Mūrcchati saṃprasaktaṃ (faints continuously), Pāṇḍu (anaemia), Kṛśa (emaciated) and Śuṣyati tṛṣṇāya (becomes dry due to thirst). This is also known as dreadful Dūṣyodara.

Plīhodara and Yakṛtodara Nidāna and Lakṣaṇās

- अशितस्यातिसङ्क्षोभाद्यानयानातिचेष्टितैः। अतिव्यवायभाराध्ववमनव्याधिकर्शनैः॥
  वामपार्श्वाश्रितः प्लीहा च्युतः स्थानात् प्रवर्धते। शोणितं वा रसादिभ्यो विवृद्धं तं विवर्धयेत्॥ (Ca.Ci.13/35-36)
- अत्याशितस्य सङ्क्षोभाद्यानयानादिचेष्टितैः॥ अतिव्यवायकर्माध्ववमनव्याधिकर्शनैः।
  वामपार्श्वाश्रितः प्लीहाच्युतः स्थानाद्विवर्द्धते॥ शोणितं वा रसादिभ्यो विवृद्धं तं विवर्द्धयेत्।

सोऽष्ठीलेवातिकठिनः प्राक् ततः कूर्मपृष्ठवत्॥ क्रमेण वर्द्धमानश्च कुक्षावुदरमावहेत्।
श्वासकासपिपासास्यवैरस्याध्मानरुग्ज्वरैः॥ पाण्डुत्व मूर्च्छाछर्दीभिर्दाहमोहैश्च संयुतम्।
अरुणाभं विवर्णं वा नीलहारिद्रराजिमत्॥ (A.H.Ni.12/22-26)

Aśitasyāti saṅkṣobhādyānayāna (travelling in the jerky vehicle immediately after intake of food), Ati ceṣṭitaiḥ (excessive physical activities), Ativyavāya (excessive sexual intercourse), Atibhāra (excessive weight lifting), Atiadhva (walking long distance), Ativamanavyādhikarśanaiḥ (emaciated due to excessive vomiting or any chronic disease), the Plīha (spleen) located in the Vāmapārśvāśrita (left side of the body) gets Cyutaḥ sthānāt (displaced from its site) and Pravardhate (enlarged) and it also gets enlarged due to Śoṇita vṛddhi (increase of blood) as a result of increase of Rasādi dhātus.

- ⊙ तस्य प्लीहा कठिनोऽष्ठीलेवादौ वर्धमानः कच्छपसंस्थान उपलभ्यते; स चोपेक्षितः क्रमेण कुक्षिं जठरमग्न्यधिष्ठानं च परिक्षिपन्नुदरमभिनिर्वर्तयति॥ (Ca.Ci.13/37)

The enlarged Plīha (spleen) is Kaṭhinoaṣṭīlevādau (hard mass similar to Dīrgho lohamaya granthi – big metal ball used by blacksmiths) in the beginning, gradually it increases and becomes like Kacchapasaṃsthāna (shape of the upper shell of tortoise). If left untreated, it covers the Kukṣi (Pārśvadeśa – lateral part of the abdomen), Jaṭhara (abdomen) and Agnyadhiṣṭhāna (seat of digestive fire) and produces the disease Udara roga.

- ⊙ दौर्बल्यारोचकाविपाकवर्चोमूत्रग्रहतमःप्रवेशपिपासाङ्गमर्दच्छर्दिमूर्च्छाङ्गसादकासश्वासमृदुज्वरानाहाग्निनाशकार्श्यास्यवैरस्यपर्वभेदकोष्ठवातशूलानि, अपि चोदरमरुणवर्णं विवर्णं वा नीलहरितहारिद्रराजिमद्भवति; एवमेव यकृदपि दक्षिणपार्श्वस्थं कुर्यात्, तुल्यहेतुलिङ्गौषधत्वात्तस्य प्लीहजठर एवावरोध इति; एतत् प्लीहोदरमिति विद्यात्॥ (Ca.Ci.13/38)

The clinical features of Plīhodara are Daurbalya (debility), Arocaka (anorexia), Avipāka (indigestion), Varcomūtragraha (retention of faeces and urine), Tamaḥpraveśa (darkness in front of the eyes), Pipāsa (thirst), Aṅgamarda (body ache), Chardi (vomiting), Mūrcchā (fainting), Aṅgasāda (tiredness of the body parts), Kāsa (cough), Śvāsa (dyspnoea), Mṛdujvara (mild fever), Ānāha (abdominal distension with constipation), Agnināśa (loss of digestive fire), Kārśya (emaciation), Āsyavairasya (abnormal taste in the mouth), Parvabheda (pain in the small joints), Koṣṭhavātaśūlāni (abdominal pain due to Vāta), Udaramaruṇavarṇa vivarṇa vā (reddish or abnormal discoloration of abdomen) and Nīlaharitahāridrarājimadbhavati (appearance of bluish, yellowish or greenish network of veins over the abdomen).

Similar are the features of Yakṛt (liver) located in the Dakṣiṇapārśva (right side of the abomen). Due to similarity in Hetu (cause), Liṅga (clinical features), Auṣadha (treatment), Yakṛtodara is included with Plīhodara.

- ⊙ उदावर्तरुजानाहैर्मोहतृड्दहनज्वरैः। गौरवारुचिकाठिन्यैर्विद्यात्तत्र मलान् क्रमात्॥ (A.H.Ni.12/27)

Vāgbhaṭa described the Plīhodara based on the doṣas; viz. Udāvarta (upward movement of Vāta), Ruja (pain), and Ānāha (abdominal distention with constipation) are caused by Vāta. Whereas Moha (delusion), Tṛḍ (thirst), Dahana (burning sensation) and Jvara (fever) are caused by Pitta, and Gaurava (heaviness), Aruci (anorexia), and Kāṭhinya (hardness of the abdomen) are caused by Kapha.

- ⊙ विदाह्यभिष्यन्दिरतस्य जन्तोः प्रदुष्टमत्यर्थमसृक् कफश्च ॥ प्लीहाभिवृद्धिं सततं करोति प्लीहोदरं तत् प्रवदन्ति तज्ज्ञाः। वामे च पार्श्वे परिवृद्धिमेति विशेषतः सीदति चातुरोऽत्र ॥ मन्दज्वराग्निः कफपित्तलिङ्गैरुपद्रुतः क्षीणबलोऽतिपाण्डुः। सव्येतरस्मिन् यकृति प्रदुष्टे ज्ञेयं यकृद्दाल्युदरं तदेव ॥ (Su.Ni.7/14-16)

Indulging in Vidāhya (food causing burning sensation during digestion) and Abhiṣyandi (channel blocking food substances) aggravates the Rakta and Kapha doṣa and produces Satataṃ Plīha vṛddhi (enlarges the spleen constantly), which is called Plīhodara by the experts. It occurs in the Vāme pārśva (left side of the abdomen) and its characteristic features are Sīdati (lassitude), Mandajvara (mild fever), Mandāgni (subdued digestive fire), Kaphapittaliṅgairupadruta (distressing symptoms of Kapha and Pitta), Kṣīṇabala (reduced strength) and Atipāṇḍu (severe anaemia). If the similar characteristic feature appears on the right side of the abdomen, it is called as Yakṛddālyudara.

Baddhagudodara Nidāna and Lakṣaṇās

- ⊙ पक्ष्मबालैः सहान्नेन भुक्तैर्बद्धायने गुदे। उदावर्तैस्तथाऽर्शोभिरन्त्रसम्मूर्च्छनेन वा॥
  अपानो मार्गसंरोधाद्धत्वाऽग्निं कुपितोऽनिलः। वर्चःपित्तकफान् रुद्ध्वा जनयत्युदरं ततः॥ (Ca.Ci.13/39-40)

Baddhāyane gude (obstruction in the passage of faeces) due to Pakṣmabālaiḥ sahānnena bhuktaiḥ (intake of food mixed with eyelashes or hair) or Udāvarta (upward movement of Vāta) or Arśobhi (pile mass) or Antrasammūrcchanena (intussusception of the intestines), leads to aggravation of Apāna Vāta due to obstruction in its passage which in turns impairs the Agni (digestive fire) and causes Varcaḥpittakaphān ruddhvā (retention of faeces, Pitta and Kapha) and produces the disease Baddhagudodara.

- ⊙ तृष्णादाहज्वरमुखतालुशोषोरुसादकासश्वासदौर्बल्यारोचकाविपाकवर्चोमूत्रसङ्गाध्मानच्छर्दिक्षवथुशिरोहृन्नाभिगुदशूलानि, अपि चोदरं मूढवातं स्थिरमरुणं नीलराजि सिरावनद्धराजिकं वा प्रायो नाभ्युपरि गोपुच्छवदभिनिर्वर्तत इति; एतद्बद्धगुदोदरमिति विद्यात्॥ (Ca.Ci.13/41)

- ⊙ पक्ष्मवालैः सहान्नेन भुक्तैर्बद्धायने गुदे॥ दुर्नामभिरुदावर्तैरन्यैर्वाऽन्त्रोपलोपिभिः।
  वर्चःपित्तकफान्रुद्ध्वाकरोतिकुपितोऽनिलः॥अपानोजठरंतेनस्युर्दाहज्वरतृट्क्षवाः।
  कासश्वासोरुसदनं शिरोहृन्नाभिपायुरुक्॥ मलसङ्गोऽरुचिश्छर्दिरुदरं मूढमारुतम्।
  स्थिरं नीलारुणसिराराजिनद्धमराजि वा॥ नाभेरुपरि च प्रायो गोपुच्छाकृति जायते। (A.H.Ni.12/28-32)

The clinical features of Baddhagudodara are Tṛṣṇā (thirst), Dāha (burning sensation), Jvara (fever), Mukhatāluśoṣa (dryness of mouth and palate), Urusāda (weakness in the thighs), Kāsa (cough), Śvāsa (dyspnoea), Daurbalya (debility), Arocaka (anorexia), Avipāka (indigestion), Varcomūtrasaṅga (retention of faeces and urine), Ādhmāna (abdominal distention), Chardi (vomiting), Kṣavathu (sneezing), Śirohṛnnābhigudaśūlāni (pain in the regions of head, cardiac, umbilical and rectum), Udaraṃ mūḍhāvātaṃ (absence of movement of Vāyu in the abdomen), Udaraṃ sthiram (motionless abdomen), Aruṇa nīlarājisirāvanaddha (appearance of reddish or bluish network of veins over the abdomen) and Rājikaṃ vā prāyo nābhyuparigopucchavadabhinirvartata (visible veins resembling the shape of cow-tail develops over the umbilical region).

- यस्यान्त्रमन्नैरुपलेपिभिर्वा बालाश्मभिर्वा सहितैः पृथग्वा । सञ्चीयते तत्र मलः सदोषः क्रमेण नाड्यामिव सङ्करो हि ॥
  निरुध्यते चास्य गुदे पुरीषं निरेति कृच्छ्रादपि चाल्पमल्पम् । हृन्नाभिमध्ये परिवृद्धिमेति त(य)च्चोदरं विट्समगन्धिकं च ॥
  प्रच्छर्दयन् बद्धगुदी विभाव्यः .... । (Su.Ni.7/17-19)

In a person, whose intestines have been stuffed with Anna (food) or Lepibhirvā (slimy) substances or Bālā (hair) or Āśma (stone) materials, the faeces along with doṣas are accumulated and gets obstructed in the rectum as like that of Nāḍyāmiva saṅkaro (dust obstructing the passage of tube) and produces Hṛnnābhi madhye parivṛddhimeti tacchodara (swelling of abdomen in between heart and umbilical region) and is associated with Kṛcchādapi cālpamalpa puriṣa nireti (little amount faeces evacuated with difficulty) and Viṭsamagandhika pracchardayan (faecal smell is present in the vomitus). This condition is known as Baddhagudodara.

Chidrodara Nidāna and Lakṣaṇās

- शर्करातृणकाष्ठास्थिकण्टकैरन्नसंयुतैः। भिद्येतान्त्रं यदा भुक्तैर्जृम्भयाऽत्यशनेन वा॥
  पाकं गच्छेद्रसस्तेभ्यश्छिद्रेभ्यः प्रस्रवद्बहिः। पूरयन् गुदमन्त्रं च जनयत्युदरं ततः॥ (Ca.Ci.13/42-43)

Śarkarātṛṇakāṣṭhāsthikaṇṭakairannasaṃyutaiḥ (intake of food mixed with gravels, straw, wood piece, bone and thorn) or Bhuktairjṛmbhayāatyaśanena (yawning after food or excessive intake of food) causes Bhidyetāntraṃ (perforation of the intestines) which gets suppurated and from these holes the Rasa prasravatbahiḥ (the watery substances comes out) and fills up the Gudamantraṃ (intestines and rectum) and produces the disease Chidrodara.

- तदधो नाभ्याः प्रायोऽभिवर्धमानमुदकोदरं भवति, यथाबलं च दोषाणां रूपाणि दर्शयति, अपि चातुरः सलोहितनीलपीतपिच्छिलकुणपगन्ध्यामवर्च उपवेशते, हिक्काश्वासकासतृष्णा प्रमेहारोचकाविपाकदौर्बल्यपरीतश्च भवति; एतच्छिद्रोदरमिति विद्यात्॥ (Ca.Ci.13/44)

- अस्थ्यादिशल्यैः सान्नैश्चद्भुक्तैरत्यशनेन वा॥ भिद्यते पच्यते वाऽन्त्रं तच्छिद्रैश्च स्रवन्बहिः।
  आम एव गुदादेति ततोऽल्पाल्पं सविड्रसः॥ तुल्यः कुणपगन्धेन पिच्छिलः पीतलोहितः।
  शेषश्चापूर्य जठरं जठरं घोरमावहेत्॥ वर्द्धयेत्तदधो नाभेराशु चैति जलात्मताम्।
  उद्रिक्तदोषरूपं च व्याप्तं च श्वासतृड्भ्रमैः॥ छिद्रोदरमिदं प्राहुः परिस्रावीति चापरे। (A.H.Ni.12/32-36)

The clinical features of Chidrodara are Adhonābhyāḥ prāyoabhivardhamānamudakodaraṃ (enlargement of abdomen especially below the umbilical region and gradually it becomes Udakodara), Yathābalaṃ ca doṣāṇāṃ rūpāṇi darśayati (shows the features of predominant doṣas), Āturaḥ Salohitanīlapītapicchilakuṇapagandhyāmavarca (the patient passes reddish, bluish, yellowish, slimy with fleshy odour and undigested faeces), Hikkā (hiccup), Śvāsa (dyspnoea), Kāsa (cough), Tṛṣṇā (thirst), Pramoha (fainting), Arocaka (anorexia), Avipāka (indigestion) and Daurbalya (debility).

- शल्यं यदन्नोपहितं तदन्त्रं भिनत्ति यस्यागतमन्यथा वा ॥ तस्मात् स्रुतोऽन्त्रात् सलिलप्रकाशः स्रावः स्रवेद्वै गुदतस्तु भूयः ।
  नाभेरधश्चोदरमेति वृद्धिं निस्तुद्यतेऽतीव विदह्यते च ॥ एतत् परिस्राव्युदरं प्रदिष्टं .... ॥ (Su.Ni.7/19-21)

Śalyaṃyadannopahitaṃ (sharp substances taken along with the food) or Yasyāgatamanyathā (otherwise lies abnormally) produces Antraṃ bhinatti (cracks in the intestines) resulting in Salilaprakāśaḥ sravaḥ sruta Sravedvai gudatastu bhūyaḥ (watery discharge comes out from the insestine and also from the anus), Nabheradhaścodarameti vṛddhi (swelling of abdomen below the umbilicus) along with Atīva nistudyate (pricking pain) and Atīva vidahyate (severe burning pain). This condition is known as Parisrāvyudara.

Udakodara Nidāna and Lakṣaṇās

- स्नेहपीतस्य मन्दाग्नेः क्षीणस्यातिकृशस्य वा। अत्यम्बुपानान्नष्टेऽग्नौ मारुतः क्लोम्नि संस्थितः॥ स्रोतःसु रुद्धमार्गेषु कफश्चोदकमूर्च्छितः। वर्धयेतां तदेवाम्बु स्वस्थानादुदराय तौ॥ (Ca.Ci.13/45-46)

- प्रवृत्तस्नेहपानादेः सहसाऽऽमाम्बुपायिनः॥ अत्यम्बुपानान्मन्दाग्नेः क्षीणस्यातिकृशस्य वा।
  रुद्ध्वाऽम्बुमार्गाननिलः कफश्च जलमूर्छितः॥ वर्धयेतां तदेवाम्बु तत्स्थानादुदराश्रितौ।
  ततः स्यादुदरं तृष्णागुदस्रुतिरुजान्वितम्॥ कासश्वासारुचियुतं नानावर्णसिराततम्।
  तोयपूर्णदृतिस्पर्शशब्दप्रक्षोभवेपथु॥ दकोदरं महत्स्निग्धं स्थिरमावृत्तनाभि तत्॥ (A.H.Ni.7/36-40)

Snehapītasya mandāgneḥ kṣīṇasyātikṛśasya vā Atyambupānan (excessive intake of water during the time of Snehapāna, subdued digestive fire, by the person suffering from debility or excessive emaciated person) leads to Naṣṭāgni (destruction of his digestive fire) and aggravation of Vāta which is situated at Kloma; obstruction of Srotas occurs after intermingling of Kapha and Udaka (water), which inturn increases the Ambu (water content) and dislodges the water from its location and produces the disease Udakodara or Jalodara.

- अनन्नकाङ्क्षापिपासागुदस्रावशूलश्वासकासदौर्बल्यानि, अपि चोदरं नानावर्णराजि सिरासन्ततमुदकपूर्णदृतिक्षोभसंस्पर्शं भवति, एतदुदकोदरमिति विद्यात्॥ (Ca.Ci.13/47)

The clinical features of Udakodara are Anannakāṅkṣā (lack of desire for food), Pipāsa (thirst), Guda srāva (anal discharge), Śūla (abdominal pain), Śvāsa (dyspnoea), Kāsa (cough), Daurbalya (debility), Udaraṃ nānāvarṇarājisirāsantataṃ (appearance of multicolored network of veins over the abdomen) and Udakapūrṇadṛtikṣobasaṃsparśa (on palpation, abdomen feels like a leather bag filled with water).

- यः स्नेहपीतोऽप्यनुवासितो वा वान्तो विरिक्तोऽप्यथवा निरूढः । पिबेज्जलं शीतलमाशु तस्य स्रोतांसि दुष्यन्ति हि तद्वहानि ।
  स्नेहोपलिप्तेष्वथवाऽपि तेषु दकोदरं पूर्ववदभ्युपैति ॥ स्निग्धं महत् सम्परिवृत्तनाभि भृशोन्नतं पूर्णमिवाम्बुना च ।
  यथा दृतिः क्षुभ्यति कम्पते च शब्दायते चापि दकोदरं तत् ॥ (Su.Ni.7/21-23)

Snehapītoapyanuvāsitovāntoviriktoapyathavā nirūḍhaḥ pibejjalaṃ śītālamāśu (one who drinks cold water soon after oleation or unctuous enema or purgation or emesis or non- unctuous enema) leads to vitiation of Udakavaha srotas and even the Srotas are Snehopalipta (lined by unctuous substances) Dakodara occurs as in the preceding one Parisrāvyudara. The characteristic features of Dakodara are Snigdha (smooth or glossy on the surface of the abdomen), Mahat samparivṛttanābhi (greatly enlarged around the umbilicus) and Bhṛśonnataṃ pūrṇamivāmbunā yathā dṛtiḥ (raised like a leather bag full with water), Kṣubhyati kampate (shakes and trembles under pressure) and Śabdāyate (makes sounds).

Avasthā bheda of Udakodara

Udakodara or Jalodara is classified into two stages namely Ajātodaka avastha and Jātodaka avastha (Picchotpatti).

Ajātodaka Avasthā Lakṣaṇās

- अजातशोथमरुणं सशब्दं नातिभारिकम्। सदा गुडगुडायच्च सिराजालगवाक्षितम्॥
  नाभिं विष्टभ्य पायौ तु वेगं कृत्वा प्रणश्यति। हृन्नाभिवङ्क्षणकटीगुदप्रत्येकशूलिनः॥
  कर्कशं सृजतो वातं नातिमन्दे च पावके। लोलस्याविरसे चास्ये मूत्रेऽल्पे संहते विषि॥ (Ca.Ci.13/55-57)

The clinical features of Ajātodaka (before accumulation of fluid in the abdomen) are Ajātaśotha (absence of swelling of the abdomen due to absence of water accumulation), Aruṇa (abdomen appears reddish in colour), Saśabda (with sound), Nātibhārika (not too heavy), Sadā guḍaguḍāyañca (persistent gurgling sound), Sirājālagavākṣitam (abdomen is covered with network of veins spreaded in shape of Gavakṣa), Nābhi viṣṭabhya pāyau tu vegaṃ kṛtvā praṇaśyati (the Vāyu generates urges to pass with distention of the umbilical region and disappears after reaching the anus), Hṛnnābhivaṅkṣaṇakaṭigudapratyeka śūlinaḥ (pain in the region of cardiac, umbilical, groin, pelvic and anus), Karkaśaṃ sṛujato vātaṃ (forceful passing of flatus), Nātimande ca pāvake (digestive fire is not much subdued), Lolasyāvirase cāsya (tastelessness due to salivation) and Mūtrealpe saṃhate viṣi (reduced urination and constipation).

- अजातोदकमित्येतैर्लिङ्गैर्विज्ञाय तत्त्वतः। उपाक्रमेद्भिषग्दोषबलकालविशेषवित्॥ (Ca.Ci.13/58)

The physician should treat this condition with due consideration of Doṣa bala (strength of the doṣa) and Kāla viśeṣā (specific season or time).

Jātodaka Avasthā (Picchotpatti) Lakṣaṇās

- तत्र अचिरोत्पन्नमनुपद्रवमनुदकमप्राप्तमुदरं त्वरमाणश्चिकित्सेत्; उपेक्षितानां ह्येषां दोषाः स्वस्थानादपवृत्ता परिपाकाद्द्रवीभूताः सन्धीन् स्रोतांसि चोपक्लेदयन्ति, स्वेदश्च बाह्येषु स्रोतःसु प्रतिहतगतिस्तिर्यगवतिष्ठमानस्त देवोदकमाप्याययति; तत्र पिच्छोत्पत्तौ मण्डलमुदरं गुरु स्तिमितमाकोठितमशब्दं मृदुस्पर्शमपगतराजीकमाक्रान्तं नाभ्यामेवोपसर्पति। ततोऽनन्तरमुदकप्रादुर्भावः। तस्य रूपाणि- कुक्षेरतिमात्रवृद्धिः, सिरान्तर्धानगमनम्, उदकपूर्णदृतिसङ्क्षोभसंस्पर्शत्वं च॥ (Ca.Ci.13/48)

- उपेक्षया च सर्वेषु दोषाः स्वस्थानतश्च्युताः॥ पाकाद्द्रवा द्रवीकुर्युः सन्धिस्रोतोमुखान्यपि।
  स्वदेश्च बाह्यस्रोतःसु विहतस्तिर्यगास्थितः॥ तदेवोदकमाप्याय्य पिच्छां कुर्यात्तदा भवेत्।
  गुरूदरं स्थिरं वृत्तमाहतं च न शब्दवत्॥ मृदु व्यपेतराजीकं नाभ्यां स्पृष्टं च सर्पति।
  तदनूदकजन्मास्मिन्कुक्षिवृद्धिस्ततोऽधिकम्॥ सिरान्तर्धानमुदकजठरोक्तं च लक्षणम्। (A.H.Ni.12/40-44)

If the Udararoga is of Acirotpanna (recent onset), Anupadrava (not associated with complications) and Anudaka (absence of fluid accumulation), then it should be treated quickly. If left untreated, the doṣas gets dislodged from its location and gets Dravībhūtā (liquified) due to Paripāka (maturation) and produces Sandhīsrotāṃsi kleda (stickiness in the joints and the srotas) and obstructs the Sveda bāhya srotas (external channels of sweat) resulting in Tiryak gati (sideways) of Sveda and it adds the quantity of fluid already accumulated in the abdomen. Thus the formation of Picchotpatti (slimy material) occurs which leads to the clinical features such as Maṇḍalamudara (abdomen become round), Guru (heaviness in the abdomen), Stimita (as if abdomen is covered with wet cloth). Ākoṭhitamaśabda (abdomen become dull on percussion), Mṛdusparśa (soft to touch), Apagatarājīkamākrāntaṃ (disappearance of network of veins), and Nābhyāmevopasarpati (primarily the disease afflicts the umbilical region and then the fluid starts to spreads) followed by Udakaprādurbhāvaḥ (water starts accumulating in the abdomen) and its signs are Kukṣīratimātravṛddhi (excessive enlargement of the sides of the abdomen), Sirāntardhānagamana (disappearance of network of veins) and Udakapūrṇadṛtisaṃkṣobha saṃsparśatvaṃ (on palpation of abdomen, one can feel fluctuation like a leather bag filled with water).

Upadrava of Udara

- तदाऽऽतुरमुपद्रवाः स्पृशन्ति छर्दतीसारतमकतृष्णाश्वासकासहिक्कादौर्बल्यपार्श्वशूलारुचि स्वरभेदमूत्रसङ्गादयः; तथाविधमचिकित्स्यं विद्यादिति॥ (Ca.Ci.13/49)

Chardi (vomiting), Atīsāra (diarrhea), Tamaka (type of dyspnoea), Tṛṣṇā (thirst), Śvāsa (dyspnoea), Kāsa (cough), Hikkā (hiccup), Daurbalya (debility), Pārśvaśūla (pain in the flanks), Aruci (anorexia), Svarabheda (hoarseness of voice) and Mūtrasaṅga (retention of urine), etc., are the Upadrava (complications) of Udara roga and such patient should not be treated as it is Asādhya.

Sādhyāsādhyata of Udara

- वातात्पित्तात्कफात् प्लीह्नः सन्निपातात्तथोदकात्। परं परं कृच्छ्रतरमुदरं भिषगादिशेत्॥
  पक्षाद्बद्धगुदं तूर्ध्वं सर्वं जातोदकं तथा। प्रायो भवत्यभावाय च्छिद्रान्त्रं चोदरं नृणाम्॥ (Ca.Ci.13/50-51)

- वातपित्तकफप्लीहसन्निपातोदकोदरम्॥ कृच्छ्रं यथोत्तरम् पक्षात्परं प्रायोऽपरे हतः।
  सर्वं च जातसलिलं रिष्टोक्तोपद्रवान्वितम्॥ (A.H.Ni.12/44-45)

Vātodara, Pittodara, Kaphodara, Plīhodara, Sannipātodara and Udakodara are the six types of Udara having increasing severity in progressive order. Baddhagudodara becomes incurable after fortnight. Both Jātodaka and Chidrodara are incurable right from the beginning.

- शूनाक्षं कुटिलोपस्थमुपक्लिन्नतनुत्वचम्। बलशोणितमांसाग्निपरिक्षीणं च वर्जयेत्॥
  श्वयथुः सर्वमर्मोत्थः श्वासो हिक्काऽरुचिः सतृट्। मूर्च्छा च्छर्दिरतीसारो निहन्त्युदरिणं नरम्॥
  जन्मनैवोदरं सर्वं प्रायः कृच्छ्रतमं मतम्। बलिनस्तदजाताम्बु यत्नसाध्यं नवोत्थितम्॥ (Ca.Ci.13/52-54)

If the patient of Udara suffers from Śūnākṣa (swollen eyes), Kuṭilopastha (curved external genitals), Klinnatanutvaca (moistening and thinning of skin), Balaśoṇitamāṃsāgniparikṣīṇaṃ (diminution of strength, blood, muscles and digestive fire), then the treatment should be avoided as it is incurable.

If the patient of Udara suffers from Śūnākṣa (swollen eyes), Kuṭilopastha (curved external genitals), Klinnatanutvaca (moistening and thinning of skin), Balaśoṇitamāṃsāgniparikṣīṇaṃ (diminution of strength, blood, muscles and digestive fire), then the treatment should be avoided as it is incurable.

If the patient of Udara suffers from Sarvamarmottha Śvayathu (swelling of all vital parts), Śvāsa (dyspnoea), Hikkā (hiccup), Aruci (anorexia), Tṛṭ (thirst), Mūrcchā (fainting), Chardi (vomiting) and Atīsāra (diarrhoea) together, it kills the patient.

Usually all types of Udara is difficult to cure right from the time of their manifestation, but if it occurs in Balavān (strong person) and in the state of Ajātāmbu (before accumulation of fluid) and Navotthita (recent onset), then can be cured with effortful treatment.

⊙ अन्ते सलिलभावं हि भजन्ते जठराणि तु । सर्वाण्येव परीपाकात्तदा तानि विवर्जयेत् ॥ (Su.Ni.7/25)

Finally, Salila (water) accumulates in the Jaṭhara (abdomen) in all kinds of Udara, and then it should be discarded from the treatment.

In Contemporary science,

Jalodara can be correlated to Ascites in contemporary science. (Please refer Topic 28)

Short Answer Questions

1. Explain Sāmānya Nidāna and Samprāpti of Udara
2. Explain Plīhodara and Yakṛtodara
3. Explain Baddhagudodara
4. Differentiate Doṣaja Udara
5. Describe Chidrodara
6. Describe Sādhyāsādhyata of Udara

Long Answer Questions

1. Define Udara, and explain the Nidāna, Samprāpti, Pūrvarūpa, Bheda and Lakṣaṇās of Udara
2. Explain the Nidāna, Lakṣaṇās and Avastha bheda of Jalodara

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TOPIC 28

Hepatobiliary Diseases

Introduction

Hepatobiliary diseases encompass a diverse array of conditions affecting the liver, gallbladder, and bile ducts. These conditions stem from various factors including viral infections, alcohol consumption, obesity, genetic predisposition, and environmental toxins. Liver-related ailments comprise hepatitis, cirrhosis, liver cancer, and fatty liver disease, while gallbladder and bile duct disorders consist of gallstones, cholecystitis, bile duct obstruction, and primary sclerosing cholangitis. This chapter will cover diseases such as cirrhosis of the liver, alcoholic and non-alcoholic liver disease, hepatitis, jaundice, and ascites.

LIVER CIRRHOSIS

Introduction

Cirrhosis of the liver, also referred to as hepatic cirrhosis and end-stage liver disease, is characterized by the degeneration of hepatic cells and liver dysfunction resulting from inflammation and damage to the liver parenchyma. The condition involves a diffuse process of fibrosis that transforms the liver architecture into structurally abnormal nodules, accompanied by hepatocellular necrosis. Cirrhosis can stem from various causes such as viral hepatitis, alcoholic liver disease, non-alcoholic steatohepatitis (NASH), autoimmune hepatitis, primary biliary cholangitis, primary sclerosing cholangitis, hemochromatosis, Wilson disease, alpha-1 antitrypsin deficiency, Budd-Chiari syndrome, drug-induced liver cirrhosis, and chronic right-sided heart failure. In 2019, the global prevalence of cirrhosis was estimated to range between 4.5% and 9%, with the estimated national prevalence in India being 2.4-4.8%.

Pathophysiology:

The pathophysiology of liver cirrhosis involves inflammation, fibrosis, and regeneration. Liver injury triggers inflammation and hepatocyte death. Activated hepatic stellate cells produce excessive extracellular matrix proteins, leading to fibrosis and scar tissue formation. As scarring progresses, the liver's structure is disrupted, impeding blood flow and causing portal hypertension. This results in splenomegaly, varices, and ascites. Despite attempts at regeneration, liver function declines, leading to impaired detoxification, protein synthesis, and bile production, potentially causing hepatic encephalopathy.

Clinical Features:

Cirrhosis of liver in early stage is asymptomatic. As the disease worsens, the symptoms like generalized fatigue, anorexia, malaise, weight loss and confused mental state may develop and the clinical signs like muscle wasting, parotid enlargement, spider naevi, bruises and purpura, loss of axillary and pubic hairs, gynaecomastia, testicular atrophy, Dupuytren's contracture, cyanosis, splenomegaly, white opaque nails with clubbing and pedal oedema may be seen.

Complications:

The complications of cirrhosis of liver are Ascites, Portal hypertension, pulmonary hypertension and hepatic encephalopathy.

Diagnosis:

Diagnosis is based on the blood tests, imaging studies and liver biopsy. Blood investigations shows elevated SGOT, SGPT, serum bilirubin, globulin and prothrombin time; elevated alkaline phosphatase, gamma-glutamyl transferase seen in biliary obstruction; decreased leucocytes, neutrophils, platelets, and albumin; Imaging studies like USG helps to assess the portal hypertension, ascites, splenomegaly and nodular liver; Endoscopy to check for abnormal veins in the esophagus or stomach; liver biopsy is done to confirm the diagnosis of cirrhosis.

Differential Diagnosis:

The differential diagnoses for cirrhosis of the liver encompass chronic hepatitis, including Viral Hepatitis (B, C, D), Autoimmune Hepatitis, and Drug-Induced Hepatitis. Additionally, Non-Alcoholic Fatty Liver Disease (NAFLD), Non-Alcoholic Steatohepatitis (NASH), Alcoholic Liver Disease, Primary Biliary Cholangitis (PBC), Primary Sclerosing Cholangitis (PSC), Hemochromatosis, Wilson's Disease, Alpha-1 Antitrypsin Deficiency, Budd-Chiari Syndrome, Congestive Hepatopathy, Sarcoidosis, Amyloidosis, Hepatocellular Carcinoma (HCC), Metastatic Liver Disease, Granulomatous Hepatitis, and Idiopathic Portal Hypertension should be considered.

ALCOHOLIC LIVER DISEASE (ALD)

Introduction

Alcoholic liver disease (ALD), also referred to as alcohol-related liver disease (ARLD), is a liver condition caused by excessive alcohol consumption. It encompasses a range of disorders commencing with alcoholic fatty liver (Hepatic steatosis), progressing to alcoholic hepatitis (Steatohepatitis), and ultimately leading to alcoholic cirrhosis, which is the most advanced and irreversible form of alcohol-related liver damage. The global prevalence of Alcoholic liver disease is approximately 4.8%, affecting 2.9% of males and 0.5% of females. In India, the prevalence of Alcoholic liver disease is 11.8%, significantly higher than the global average.

Pathophysiology:

The pathophysiology of Alcoholic Liver Disease involves a complex interplay of (acetaldehyde toxicity, oxidative stress, inflammation, and fibrosis) mechanisms triggered by chronic alcohol consumption. Hepatocyte metabolism of alcohol gives rise to acetaldehyde, a toxic intermediate that damages liver cells. Acetaldehyde induces modifications in cellular membranes, promotes lipid peroxidation, and disrupts mitochondrial function. Chronic alcohol exposure also induces the upregulation of cytochrome P450 2E1 (CYP2E1), intensifying oxidative stress and exacerbating liver injury. Alcohol-induced oxidative stress triggers inflammation, activating Kupffer cells and hepatic stellate cells (HSCs). Activated HSCs produce collagen, leading to fibrosis and scar tissue formation. As fibrosis a progress, liver architecture is disrupted, impeding blood flow and causing portal hypertension. This results in splenomegaly, varices, and ascites. Prolonged alcohol consumption also impedes hepatic regeneration, exacerbating the extent of damage. Disrupted cytokine balance, particularly diminished IL-10 and increased TNF-α, sustains the inflammatory process.

Clinical features:

Alcoholic Liver Disease (ALD) progresses through three distinct stages: Alcoholic Fatty Liver (Hepatic Steatosis), Alcoholic Hepatitis, and Alcoholic Cirrhosis.

Initially, **Hepatic Steatosis** typically manifests asymptomatically with the accumulation of fat in the liver. In certain cases, the liver may exhibit enlargement and a smooth appearance. Subsequently, **Alcoholic Hepatitis** ensues, characterized by inflammation, fatigue, fever, jaundice, right upper quadrant pain, and tender hepatomegaly. If left untreated, **Alcoholic Cirrhosis** develops, marked by irreversible liver damage, complications associated with cirrhosis, and portal hypertension, including oesophageal varices, upper gastrointestinal bleeding, splenomegaly, ascites, and portosystemic encephalopathy.

Furthermore, patients with ALD may manifest extrahepatic symptoms such as Dupuytren contracture, vascular spiders, myopathy, peripheral neuropathy, hypogonadism, and feminization. Undernutrition can lead to multiple vitamin deficiencies, enlarged parotid glands, and leukonychia. Thiamine deficiency may precipitate Wernicke encephalopathy and Korsakoff psychosis. Pancreatitis and Hepatitis C commonly coexist, exacerbating the progression of liver disease. Zieve syndrome, characterized by hyperlipidemia, hemolytic anemia, and jaundice, is rarely encountered in patients.

Diagnosis

The diagnosis of alcoholic liver disease relies on the patient's alcohol consumption history, liver function tests, complete blood count (CBC), and liver biopsy. Liver function tests commonly reveal a notable elevation in SGOT/AST (serum glutamic oxaloacetic transaminase/ aspartate aminotransferase) in contrast to SGPT/ALT (serum glutamic-pyruvic transaminase/ alanine aminotransferase) levels. Additionally, characteristic indicators include hypoalbuminemia, hyperbilirubinemia, hypertriglyceridemia, and elevated GGTP (gamma-glutamyl transpeptidase). A complete blood count is essential to exclude infection and to assess potential complications such as anaemia, thrombocytopenia, and leukemoid reaction in alcoholic hepatitis. In cases where diagnostic uncertainty exists, a liver biopsy may provide a definitive diagnosis.

NON-ALCOHOLIC LIVER DISEASE

Introduction

Non-alcoholic liver disease, also known as Non-alcoholic fatty liver disease (NAFLD), is a condition characterized by the accumulation of fat in the liver, closely associated with metabolic disorders, including central obesity, dyslipidemia, hypertension, hyperglycemia, and persistent abnormalities of liver function tests. Non-alcoholic fatty liver disease (NAFLD) encompasses two main subtypes, namely, Non-alcoholic fatty liver (NAFL) and Non-alcoholic steatohepatitis (NASH).

NAFLD affects a significant portion of the global population, with an estimated prevalence of 32.4% among adults worldwide. The incidence of NAFLD is increasing in tandem with the rising rates of obesity and type 2 diabetes. In India, NAFLD prevalence ranges from 9.7% to 32.7%, varying across regions and populations. Approximately 30-40% of Indian adults with obesity or type 2 diabetes are estimated to have NAFLD. The Indian subcontinent's genetic predisposition, dietary habits, and socioeconomic factors contribute to this high prevalence.

Pathophysiology:

Non-Alcoholic Fatty Liver Disease (NAFLD) involves a complex interplay of metabolic, hormonal, and inflammatory mechanisms. It is characterized by excessive hepatic lipid accumulation, primarily triglycerides, due to an imbalance between lipid influx, synthesis, export, and oxidation. Insulin resistance, often associated with obesity and type 2 diabetes, plays a pivotal role in NAFLD development. Impaired insulin signalling promotes release of free fatty acids into the bloodstream, taken up by hepatocytes, leading to increased triglyceride synthesis and reduced lipid export. Mitochondrial dysfunction and impaired beta-oxidation further contribute to lipid accumulation. Inflammatory processes promote the progression from simple steatosis to non-alcoholic steatohepatitis (NASH), characterized by hepatocellular injury and fibrosis. Advanced NAFLD stages may lead to cirrhosis and potentially hepatocellular carcinoma. Genetic predisposition, dietary factors, and gut microbiome alterations also modulate NAFLD pathophysiology.

Clinical Features:

Non-alcoholic fatty liver disease (NAFLD) typically presents with no symptoms, although some individuals may experience fatigue, right upper quadrant abdominal pain, thirst, bloating, sleep disturbances, hepatomegaly, acanthosis nigricans, and lipomatosis. In contrast, those who progress to non-alcoholic steatohepatitis (NASH)-associated cirrhosis, end-stage liver disease, or hepatocellular carcinoma (HCC) often exhibit more severe symptoms, including nausea, vomiting, jaundice, pruritus, ascites, memory impairment, easy bleeding, and loss of appetite. The most common clinical sign of NAFLD is mild to moderate hepatomegaly. Advanced stages of the disease spectrum may manifest signs of end-stage liver disease, such as jaundice, spider angiomas, palmar erythema, caput medusa, gynecomastia, Dupuytren's contracture, ascites, and petechiae.

Diagnosis:

Diagnosis is based on liver function tests, imaging studies, and liver biopsy. Liver function tests may demonstrate mildly elevated serum aminotransferases, whereas the majority of patients exhibit normal liver enzyme levels. The ratio of aspartate aminotransferase (AST) to alanine aminotransferase (ALT) typically falls below 1. Elevated gamma-glutamyl transferase (GGT) in NAFLD may serve as an indicator of increased mortality and disease progression. Advanced NAFLD may manifest with hypoalbuminemia, hyperbilirubinemia, thrombocytopenia, and prolonged prothrombin time attributable to hepatic synthetic dysfunction. While ultrasound imaging represents a routine method for assessing fatty liver, a liver biopsy stands as the definitive means for diagnosing NAFLD.

Differential Diagnosis:

Alcoholic Liver Disease (ALD) and Non-Alcoholic Fatty Liver Disease (NAFLD) require a thorough differential diagnosis to exclude other liver conditions. Key considerations encompass Non-Alcoholic Fatty Liver Disease (NAFLD), Viral Hepatitis (B and C), Autoimmune Hepatitis, Wilson's disease, Hemochromatosis, and Alpha-1 Antitrypsin Deficiency. Additionally, potential diseases for consideration include Pancreatitis, Cardiac failure, Malignancies (e.g., hepatocellular carcinoma), Infections (e.g., tuberculosis), Drug-induced liver injury, Toxic liver damage, Budd-Chiari syndrome, and Portal vein thrombosis.

HEPATITIS

Introduction

Hepatitis, characterized by liver inflammation, can be caused by acute or chronic viral infections, including hepatitis A, B, C, D, and E, as well as non-viral factors. Non-viral hepatitis encompasses various etiologies such as drug/toxin-induced liver injury, alcoholic liver disease, autoimmune hepatitis, non-alcoholic steatohepatitis (NASH), and ischemic hepatitis. According to the World Health Organization (WHO), approximately 296 million people worldwide live with chronic hepatitis B, and 58 million with chronic hepatitis C. In India, an estimated 40 million individuals are affected by chronic hepatitis B, and 6-12 million by chronic hepatitis C.

- **Acute or chronic viral hepatitis** is an inflammation of liver caused by specific group of viruses such as Hepatitis A, B, C, D and E, having affinity towards liver cells and producing typical clinical, biochemical, immunological and morphological features. All these viruses produce almost similar clinical features but it can be differentiated by their antigen characteristics.
  - **Hepatitis A (HAV) :** Hepatitis A is the commonest type of viral hepatitis and it is caused by a single stranded RNA enterovirus mainly affecting the children and young adults through the faeco–oral route. Its incubation period is 15-45 days. Children are asymptomatic but in young adults the symptoms like fever, nausea, vomiting, diarrhoea, loss of appetite, headache, stomach pain, jaundice and weakness occur. Diagnosis is based on the presence of IgM anti- HAV which suggests acute infection and IgG anti-HAV which suggests previous infection or vaccine acquired immunity; treatment includes symptomatic and supportive measures. Prevention is by Hepatitis A vaccination.
  - **Hepatitis B (HBV) :** Hepatitis B is caused by a DNA virus and it replicates within the nuclei of infected hepatocytes. HBV spreads through parenteral route i.e. transfusion of infected blood and blood products, intravenous drug use, sexual contact, use of contaminated syringes, needles etc. and it may also occur to newborn from mother. Incubation period of HBV is ranging from 6 weeks to 6 months. The clinical features are fever, nausea, vomiting, weakness, diarrhoea, loss of appetite, headache, stomach pain, followed by jaundice and urticaria and arthralgia occasionally occurs. In acute cases, it produces fulminant hepatitis and death may occur; in chronic cases, cirrhosis and hepatocellular carcinoma can develop. Diagnosis is based on the presence of Hepatitis B surface antigen (HBsAg) which suggests positive acute infection. To determine the prognosis of HBV, Hepatitis B e antigen (HBeAg) and antibody to hepatitis B e antigen (anti-HBe) is done. Treatment includes supportive measures, use of oral anti-viral drugs like Lamivudine, Telbivudine or Entecavir and liver transplantation. Prevention is by Hepatitis B vaccination.

  - **Hepatitis C (HCV)** is caused by an RNA virus which spreads through parenteral route by needles, tattoos or body piercing. Sexual and perinatal transmission from mother to infant is uncommon. Incubation period ranges from 15-60 days. In chronic hepatitis, cirrhosis and rarely hepatocellular carcinoma develops and diagnosis depends on the detection of anti-HCV antibodies. Treatment includes supportive care and use of anti-viral drugs. No vaccine is available.
  - **Hepatitis D (HDV)** is a delta hepatitis virus caused by a defective RNA virus which requires HBV for its replication and it causes acute hepatitis only in the presence of hepatitis B viral infection or chronic carriers of hepatitis B. It is transmitted through parenteral route in drug addicts and during blood transfusion. Diagnosis depends on the demonstration of anti HDV IgM antibodies; presence of HbsAg and IgM anti hepatitis B core (IgM anti-HBc) which suggests co-infection. Treatment includes supportive care and use of drug Interferon alpha.
  - **Hepatitis E (HEV)** is caused by a RNA virus and it spreads through oral route. Fulminant hepatitis and death are common especially in pregnant women. Diagnosis is made by the detection of HEV antibodies and presence of IgM antibodies against HEV which suggests the recent infection. Treatment includes symptomatic approach, supportive care and use of antiviral drugs like Ribavirin.
- **Chronic non-viral hepatitis** is caused by the use of drugs/toxins, alcoholic hepatitis and autoimmune hepatitis, non alcoholic steatohepatitis and ischemic hepatitis. Drug/toxin induced hepatitis is caused by chemical agents, toxins and medications resulting in liver injury; alcoholic hepatitis is caused by excessive consumption of alcohol resulting in liver failure; autoimmune hepatitis may be caused by an abnormal immune response against liver cells; non-alcoholic steatohepatitis (NASH) is caused by metabolic syndrome, obesity, insulin resistance diabetes resulting in liver cell death, liver inflammation and possible fibrosis; ischemic hepatitis is caused by reduced blood flow to the liver due to shock, heart failure, or vascular insufficiency.

Clinical features:

Chronic hepatitis is often asymptomatic. As the disease progresses, patient may present with symptoms like fatigue, nausea, vomiting, poor appetite and joint pain associated with acne, amenorrhea, hirsutism, ulcerative colitis, pulmonary fibrosis, thyroiditis, nephritis and haemolytic anaemia. In advanced stage of disease, patient may develop cirrhosis of liver with jaundice, weight loss, ascites, oedema and also in some patients complications like portal hypertension, hepatic encephalopathy and liver cancer may develop.

Diagnosis:

Diagnosis is based on the blood tests, imaging studies and liver biopsy. Blood investigations show elevated SGOT and SGPT in almost all types of hepatitis. But the degree of elevation, the predominance of SGOT and SGPT elevation, and the ratio between SGOT and SGPT are helpful for the diagnosis. For example, SGOT is grossly elevated in alcoholic hepatitis; SGPT is grossly elevated in ischemic hepatitis. Imaging studies like USG, CT and MRI are helps to diagnose the fatty liver tissue and cirrhosis; liver biopsy is helps to assess the inflammation and fibrosis of the liver.

Differential Diagnosis:

Hepatitis, inflammation of the liver, requires a comprehensive differential diagnosis to distinguish between various causes. Viral hepatitis (A, B, C, D, and E), autoimmune hepatitis, alcoholic hepatitis, non-alcoholic fatty liver disease (NAFLD), and drug-induced liver injury are primary considerations. Other potential causes include bacterial infections, parasitic infections, fungal infections, toxin-induced liver damage, Wilson's disease, hemochromatosis, and alpha-1 antitrypsin deficiency.

JAUNDICE

Introduction

Jaundice, also known as hyperbilirubinemia, is characterized by the yellowish discoloration of the skin, mucous membranes, and deeper tissues resulting from the accumulation of excess bilirubin. The average level of bilirubin in the blood ranges from 0.8 to 1.2 mg/dL, and jaundice occurs when bilirubin exceeds 2 mg/dL. Approximately 170 million people worldwide suffer from liver diseases, with jaundice being a common symptom. In India, 2.5 million new cases of jaundice are reported annually, and an estimated 40 million people live with chronic hepatitis B and 6 to 12 million with chronic hepatitis C.

Pathophysiology:

Hyperbilirubinemia is classified into conjugated and unconjugated hyperbilirubinemia. The manifestation of jaundice arises from impaired bilirubin excretion. Unconjugated bilirubin, also known as indirect bilirubin or free bilirubin, is a byproduct of the breakdown of deteriorating red blood cells, predominantly derived from hemoglobin. This process encompasses:

- ⊙ Degradation of hemoglobin into iron and biliverdin, a green pigment.
- ⊙ Conversion of biliverdin into unconjugated bilirubin.

Unconjugated bilirubin, being lipid-soluble, binds to albumin and is transported through the bloodstream to the liver. Within the liver, unconjugated bilirubin undergoes glucuronidation to form conjugated bilirubin, also referred to as direct bilirubin, rendering it water-soluble. Subsequently, conjugated bilirubin is excreted through bile into the duodenum.

In the intestinal tract, bilirubin is metabolized by bacteria to generate urobilinogen. A portion of urobilinogen is further converted to stercobilin, which imparts a dark brown color to feces. The residual urobilinogen is reabsorbed by intestinal cells, transported to the kidneys via the bloodstream, and subsequently excreted in the urine.

Classification:

Jaundice is classified into three types namely, Pre hepatic, Hepatic and Post hepatic jaundice.

- ⊙ **Pre hepatic Jaundice** is also known as haemolytic jaundice, where there will be excessive destruction of red blood cells in the reticulo-endothelial system resulting in excessive production of unconjugated bilirubin from haemoglobin For example Haemolytic anaemia, Thalassemia and Sickle cell anaemia etc. and it is characterised by anaemia, fever, mild yellow discoloration of skin, splenomegaly and moderately enlarged liver with increased indirect or unconjugated bilirubin.
- ⊙ **Hepatic jaundice** is also known as hepato cellular jaundice, where there will be damage of parenchymal liver cells due to infection, inflammation, drug induced, toxins and rarely genetic diseases. For example Hepatitis, Gilbert's syndrome, alcoholic liver disease etc. and it is characterised by low grade fever, malaise, anorexia, nausea, vomiting, mild itching sensation, recurrent abdominal pain, orange tinged discoloration of the skin, enlarged liver and spleen may or may not be enlarged and associated with increased level of indirect and direct bilirubin.
- ⊙ **Post hepatic jaundice** is also known as obstructive jaundice, where there will be obstruction of the biliary tract resulting in decreased excretion of conjugated bilirubin. For example, Choledocholithiasis, Carcinoma of liver, head of pancreas and stomach etc. and it is characterised by recurrent abdominal pain, severe itching sensation, asthenia, weight loss, wasting, bruises, yellowish green discoloration of the skin, enlarged liver, palpable gall bladder and is associated with increased direct bilirubin.

Diagnosis

Diagnosis is based upon the blood investigations which shows elevated alkaline phosphatase and grossly elevated SGOPT and SGPT levels in Hepatic jaundice; increased SGOT and SGPT levels and grossly elevated alkaline phosphatase level in obstructive jaundice. Stool examination shows slightly pale colour in hepatic jaundice whereas clay colour in obstructive jaundice; in urine, urobilinogen is absent in obstructive, excess in both pre hepatic and hepatic jaundice; bilirubin is absent in pre hepatic and excess in hepatic and obstructive jaundice. Radiology and ultrasonography may be helpful in obstructive jaundice to rule out gall stones, carcinoma etc.

Differential Diagnosis:

The differential diagnosis of jaundice can be categorized into prehepatic, hepatic, and posthepatic causes

- **Pre-hepatic (haemolytic):** Haemolytic anaemia, polycythemia vera, and haemolysis.
- **Hepatic (liver cell damage):** Viral hepatitis, alcoholic liver disease, NAFLD, autoimmune hepatitis, and liver cirrhosis.
- **Post-hepatic (obstructive):** Biliary obstruction, choledocholithiasis, primary sclerosing cholangitis, and pancreatic cancer.

ASCITES

Introduction

Ascites is a pathological condition characterized by the accumulation of fluid within the peritoneal cavity. It is primarily associated with liver diseases such as cirrhosis, alcoholic hepatitis, and hepatic vein obstruction. Other etiologies include cardiac failure, nephrotic syndrome, malignancies, heart failure, tuberculosis, and pancreatitis. Globally, liver cirrhosis constitutes 75-80% of ascites cases, followed by malignancies (10-15%) and heart failure (5-10%). In India, an estimated 2.5-5 million individuals are afflicted with ascites, with liver cirrhosis being the predominant cause (60-70%), followed by malignancies (15-20%), tuberculosis, and heart failure.

Pathophysiology:

The pathophysiology of ascites is the result of a multifaceted interplay of factors. The primary mechanism involves portal hypertension, wherein elevated pressure in the portal vein induces the leakage of fluid from blood vessels into the peritoneal space. This phenomenon is frequently attributed to liver cirrhosis, which gives rise to scarring and fibrosis, impeding blood flow. As portal pressure escalates, hydrostatic forces expel fluid from blood vessels, while diminished oncotic pressure, stemming from hypoalbuminemia, hampers fluid reabsorption.

Additional pivotal factors contribute to ascites formation. Splanchnic vasodilation, modulated by nitric oxide and other vasodilators, augments blood flow to the gut, resulting in fluid accumulation. Renal vasoconstriction and activation of the renin-angiotensin-aldosterone system (RAAS) promote sodium retention, exacerbating fluid buildup. Increased permeability of blood vessels and lymphatic vessels facilitates fluid leakage into the peritoneal cavity.

Systemic and local factors sustain ascites. Systemic inflammation, oxidative stress, and cytokine release contribute to vascular dysfunction. Locally, peritoneal lymphatic dysfunction and fibrosis impede fluid drainage. The accumulation of fluid in the peritoneal cavity increases intra-abdominal pressure, compromising organ function and predisposing individuals to complications such as spontaneous bacterial peritonitis and hepatorenal syndrome.

Clinical features:

Mild ascites causes no symptoms. Moderate ascites causes painless progressive abdominal distension associated with weight gain and increased abdominal girth. Severe ascites causes abdominal pain or discomfort with abdominal distension along with dyspnoea due to fluid accumulation and increased abdominal pressure. Symptoms of spontaneous bacterial peritonitis include fever, abdominal tenderness and confusion.

Diagnosis:

Diagnosis is based on physical examination, imaging studies and serum-ascites albumin gradient (SAAG) level. Physical examination shows the presence of shifting dullness, fluid thrill and everted umbilicus. Other signs include spider naevi, jaundice, palmar erythema; muscle wasting, gynecomastia and leukonychia are seen in ascites due to severe liver diseases. Jugular venous distension, pulmonary congestion and peripheral oedema are seen in ascites due to cardiac failure. Imaging studies such as USG and CT abdomen reveals the fluid volume even less than 100mL. Diagnostic abdominal paracentesis is done with ascitic fluid to evaluate the cause of the ascites.

Serum-ascites albumin gradient (SAAG) high level (>1.1g/dL) indicates the ascites due to portal hypertension and it is seen in cirrhosis, alcoholic hepatitis, heart failure, massive hepatic metastases, heart failure/pericarditis, Budd-Chiari syndrome, portal vein thrombosis, and idiopathic portal fibrosis. SAAG low level (<1.1g/dL) indicates the ascites due to peritoneal carcinomatosis, peritoneal tuberculosis, pancreatitis, serositis and nephrotic syndrome without portal hypertension.

Differential Diagnosis:

Ascites necessitates a thorough differential diagnosis, which includes liver-related conditions (cirrhosis, liver cancer, and hepatic vein thrombosis), cardiac issues (heart failure, constrictive pericarditis, and cardiac tamponade), malignancy-related ailments (ovarian, pancreatic, and gastrointestinal cancers), infection-related illnesses (spontaneous bacterial peritonitis and tuberculosis), and other potential causes (pancreatic disorders, renal disorders, vasculitis, and drug-induced conditions).

MCQ's

1. Which of the following is NOT a common cause of liver cirrhosis

a) Viral hepatitis
b) Alcoholic liver disease
c) Non-alcoholic steatohepatitis (NASH)
d) Acute pancreatitis

2. What is the primary mechanism leading to the formation of ascites in cirrhosis
   a) Increased oncotic pressure
   b) Portal hypertension
   c) Decreased hydrostatic pressure
   d) Increased renal blood flow
3. Which of the following clinical features is NOT typically associated with early-stage cirrhosis
   a) Generalized fatigue
   b) Anorexia
   c) Muscle wasting
   d) Weight loss
4. Which diagnostic test is used to confirm the diagnosis of cirrhosis
   a) Blood tests
   b) Imaging studies
   c) Endoscopy
   d) Liver biopsy
5. Which complication of cirrhosis involves increased pressure in the portal vein
   a) Ascites
   b) Portal hypertension
   c) Pulmonary hypertension
   d) Hepatic encephalopathy
6. Which of the following is the most advanced and irreversible form of Alcoholic Liver Disease
   a) Alcoholic fatty liver (Hepatic steatosis)
   b) Alcoholic hepatitis (Steatohepatitis)
   c) Alcoholic cirrhosis
   d) Non-alcoholic steatohepatitis (NASH)
7. Which toxic intermediate is produced during the metabolism of alcohol in hepatocytes
   a) Acetone
   b) Acetaldehyde
   c) Ethanol
   d) Methanol
8. Which enzyme's upregulation intensifies oxidative stress in Alcoholic Liver Disease
   a) Cytochrome P450 2E1 (CYP2E1)
   b) Alcohol dehydrogenase (ADH)
   c) Aldehyde dehydrogenase (ALDH)
   d) Glutathione peroxidase (GPx)
9. Which of the following is NOT a clinical feature of Alcoholic Hepatitis
   a) Fatigue
   b) Jaundice
   c) Right upper quadrant pain
   d) Hepatic encephalopathy

10. What is a characteristic indicator of alcoholic liver disease in liver function tests
    a) Elevated SGOT/AST compared to SGPT/ALT
    b) Decreased bilirubin levels
    c) Low GGTP levels
    d) High albumin levels
11. Which of the following metabolic disorders is closely associated with NAFLD
    a) Central obesity
    b) Dyslipidemia
    c) Hypertension
    d) All of the above
12. What are the two main subtypes of NAFLD
    a) Alcoholic fatty liver and alcoholic hepatitis
    b) Non-alcoholic fatty liver (NAFL) and non-alcoholic steatohepatitis (NASH)
    c) Hepatic steatosis and hepatic fibrosis
    d) Cirrhosis and hepatocellular carcinoma
13. Which factor plays a pivotal role in the development of NAFLD
    a) Insulin resistance
    b) Viral infection
    c) Genetic mutation
    d) Autoimmune response
14. Which clinical feature is most commonly associated with NAFLD
    a) Severe jaundice
    b) Mild to moderate hepatomegaly
    c) Severe abdominal pain
    d) Frequent vomiting
15. What is the definitive means for diagnosing NAFLD
    a) Liver function tests
    b) Imaging studies
    c) Liver biopsy
    d) Blood glucose levels
16. What is the incubation period for Hepatitis A
    a) 6 weeks to 6 months
    b) 15-45 days
    c) 15-60 days
    d) 1-2 weeks
17. Which hepatitis virus requires Hepatitis B virus for its replication
    a) Hepatitis A
    b) Hepatitis C
    c) Hepatitis D
    d) Hepatitis E

18. Which diagnostic marker indicates an acute Hepatitis B infection
a) IgM anti-HAV
b) Hepatitis B surface antigen (HBsAg)
c) Anti-HCV antibodies
d) IgM anti-HDV

19. What is the primary route of transmission for Hepatitis C
a) Faeco-oral route
b) Parenteral route
c) Sexual contact
d) Perinatal transmission

20. Which hepatitis virus is most commonly associated with fulminant hepatitis and death
a) Hepatitis A
b) Hepatitis B
c) Hepatitis C
d) Hepatitis E

21. What is the average level of bilirubin in the blood
a) 0.8 to 1.2 mg/dL
b) 1.5 to 2.0 mg/dL
c) 2.5 to 3.0 mg/dL
d) 3.5 to 4.0 mg/dL

22. At what bilirubin level does jaundice typically occur
a) 1 mg/dL
b) 2 mg/dL
c) 3 mg/dL
d) 4 mg/dL

23. Which type of jaundice is also known as haemolytic jaundice
a) Pre hepatic jaundice
b) Hepatic jaundice
c) Post hepatic jaundice
d) Neonatal jaundice

24. What is the primary cause of hepatic jaundice
a) Excessive destruction of red blood cells
b) Obstruction of the biliary tract
c) Damage to parenchymal liver cells
d) Genetic disorders

25. Which diagnostic marker is elevated in obstructive jaundice
a) SGOT
b) SGPT
c) Alkaline phosphatase
d) Bilirubin

26. What color is the stool in obstructive jaundice
a) Dark brown
b) Slightly pale
c) Clay-colored
d) Yellowish-green

27. What is the primary cause of ascites globally
a) Malignancies
b) Heart failure
c) Liver cirrhosis
d) Tuberculosis

28. Which factor primarily contributes to the formation of ascites in liver cirrhosis
a) Decreased hydrostatic pressure
b) Portal hypertension
c) Increased oncotic pressure
d) Renal vasodilation

29. What is a common clinical feature of moderate ascites
a) Severe abdominal pain
b) Painless progressive abdominal distension
c) Jaundice
d) Dyspnoea

30. Which diagnostic marker indicates ascites due to portal hypertension
a) Low SAAG (<1.1 g/dL)
b) High SAAG (>1.1 g/dL)
c) Elevated SGOT
d) Elevated SGPT

Answer Keys

1	d	2	b	3	c	4	d	5	b	6	c	7	b	8	a	9	d	10	a
11	d	12	b	13	a	14	b	15	c	16	b	17	c	18	b	19	b	20	d
21	a	22	b	23	a	24	c	25	c	26	c	27	c	28	b	29	b	30	b

Short Answer Questions

1. Explain Cirrhosis of Liver
2. Explain Alcoholic liver disease
3. Explain Non- Alcoholic liver disease
4. Explain Hepatitis in detail
5. Describe Jaundice
6. Describe Ascites.

◆

TOPIC 29

Kuṣṭha

Introduction

Kuṣṭha is one among the Aṣṭa mahāgada (eight severe diseases) and a Santarpaṇotha vyādhi (caused by over satiation). It is considered as Dīrgha roga (chronic disease) and Kaṣṭhataro roga (more distressing disease) because if the person dies due to Kuṣṭha, it appears even in the rebirth of the person. Ācārya Caraka considered Kuṣṭha as one of the Aupasargika roga (contagious disease) which may spread by contact. Ācārya Suśruta stated Kṛmi (helminthiasis) as one of the causative factors of Sarva Kuṣṭha (all types of Kuṣṭha).

In Vācaspatyam, Kuṣṭha is considered as Mahāpātaka (heinous sin) and it persists for Saptajanma jāyate (up to seven births) in the form of Vyādhi rūpa bādhate (disease) and it is Kṛcchrādhibhi (difficult to cure). Hence, in such diseases, Prāyaścita (atonement) should be carried out. Tvakdoṣa, Tvak roga, Tvakāmaya and Carma roga are the terms mentioned as synonyms of Kuṣṭha and it specifically causes Sparśanaghna (impairment of tactile sensation).

Vyutpatti

- कुष् + हनि कुषीति । इति क्थन् । कुष्ठं । (SKD)
- कुष् निष्कर्षे + हनि कुषीति । इति क्थन् । कुष्ठं । (SKD)

The word 'Kuṣṭha' is derived from 'Kuṣ niṣkarṣane' dhātu by adding 'Kthan' pratyaya. The word 'Kuṣ' means to extract or to destroy or to tear or to draw out or to deform and the word 'Kthan' means certainty. Hence, the word 'Kuṣṭha' means that which destroys with certainty.

Nirūkti and Paribhāṣa

- कुष्ठं, कुष्णाति रोगम् । (SKD)

That which causes despise or contemptible situations.

- कुष्ठं, कुष्णाति शरीरस्थशोणितं विकुरुते इति । (SKD)

The aggravated Rakta leads to destruction of body, so it is called Kuṣṭha

- कालेनोपेक्षितं यस्मात्सर्वं कुष्णाति तद्वपुः। (A.H.Ni.14/4)

That which disfigures the body in the course of time is called Kuṣṭha.

- त्वचः कुर्वन्ति वैवर्ण्यं दुष्टाः कुष्ठमुशन्ति तत्॥ (A.H.Ni.14/3)

That which produces discoloration of the Tvak (skin) is known as Kuṣṭha.

- कृष्णाति तस्मात्तत् कुष्ठमित्युच्यते। (Aru.A.H.Ni.14/4)

That which destroys the body is called Kuṣṭha.

- त्वचागः सर्पति रोगदोषः कुष्ठेति संज्ञा प्रवदन्ति धीराः॥ (Hā.Sa.Tṛ.39/2)

The aggravated doṣa gets located in the skin and spreads throughout the body, which is called Kuṣṭha by an intelligent physician.

- दोषाणां संञ्चितानां तु त्वङ्मांसाच्छास्रचारिणाम् । प्रदूषणं हि सर्वेषां कुष्ठमित्यभिधीयते ॥ (Bhe.Sa.Ci.6/3)

The aggravated three doṣas move to the Tvak (skin), Māṃsa (muscles), Acchā (lymph) and Asṛk (blood) and vitiate all of these and produce the disease called Kuṣṭha.

Sāmānya Nidāna of Kuṣṭha

- विरोधीन्यन्नपानानि द्रवस्निग्धगुरूणि च। भजतामागतां छर्दिं वेगांश्चान्यान्प्रतिघ्नताम्॥
  व्यायाममतिसन्तापमतिभुक्त्वोपसेविनाम्। शीतोष्णलङ्घनाहारान्क्रमंमुक्त्वानिषेविणाम्॥
  घर्मश्रमभयार्तानां द्रुतं शीताम्बुसेविनाम्। अजीर्णाध्यशिनां चैव पञ्चकर्मापचारिणाम्॥
  नवान्नदधिमत्स्यातिलवणाम्लनिषेविणाम्। माषमूलकपिष्टान्नतिलक्षीरगुडाशिनाम्॥
  व्यवायं चाप्यजीर्णेऽन्ने निद्रां च भजतां दिवा। विप्रान् गुरून् धर्षयतां पापं कर्म च कुर्वताम्॥ (Ca.Ci.7/4-8)

The causative factors of Kuṣṭha are:

- Virodhīnyannapāna (incompatible diet and drinks)
- Dravasnigdhagurūṇi (excessive intake of liquids, unctuous and heavy food substances);
- Bhajatāmāgatāṃ chardi vegaṃścsnyānpratighnatām (suppression of urge of vomiting after eating food and also suppression of other urges);
- Vyāyāmāmatisantāpamatibhuktvopasevinām (indulging in excessive exercise or exposure to excessive heat after taking excessive quantity of food);
- Śītoṣṇa (use of cold substances followed by hot substances);
- Laṅghanāharān kramaṃ bhuktvāniṣeviṇām (fasting or eating frequently and not following the method of taking food);
- Dharmaśramabhayārtānāṃ drutaṃ śītambusevinām (drinking cold water immediately after exposure to sun, exertion and fear);

- Ajīrṇādhyaśinā̄ṃ (intake of food during indigestion or before digestion of previous meals);
- Pañcakarmāpacāriṇām (improper management during five purificatory therapies);
- Excessive intake of Navānna (recently harvested grains), Dadhi (curd), and Matsya (fish);
- Atilavaṇamlaniṣeviṇām (excessive intake of saline and sour substances);
- Excessive consuming of Maṣā (black gram), Mūlaka (radish), Piṣṭānna (paste of pulses), Tila (sesame), Kṣīra (milk) and Guḍa (jaggery);
- Vyavāyaṃ cāpyājīrṇe anne (indulging in sexual intercourse during indigestion);
- Divāsvapna (day sleep);
- Viprān gurūn dharṣayatāṃ (disrespect to brāḥmaṇās and teachers) and
- Pāpa karma (doing sinful acts)

Saṃprāpti of Kuṣṭha

- वातादयस्त्रयो दुष्टास्त्वग्रक्तं मांसमम्बु च। दूषयन्ति स कुष्ठानां सप्तको द्रव्यसङ्ग्रहः॥ अतः कुष्ठानि जायन्ते सप्त चैकादशैव च। न चैकदोषजं किञ्चित् कुष्ठं समुपलभ्यते॥ (Ca.Ci.7/9-10)

The aggravated Vātadi doṣas vitiate the Tvak (skin), Rakta (blood), Māṃsa (muscles) and Ambu (lymph). Thus these seven pathogenic substrata of Kuṣṭha produce seven types of Mahā Kuṣṭha and eleven types of Kṣudra Kuṣṭha. Single doṣa cannot cause a single Kuṣṭha on its own.

Saṃprāpti Ghaṭaka

Doṣa	Tridoṣa
Dūṣya	Tvak, Rasa, Māṃsa, Lasīkā
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhya janya Āma
Srotas	Rasa, Rakta, Māṃsa, and Sveda vaha
Srotoduṣṭi	Saṅga, Vimārga gamana
Adhiṣṭhāna	Tvak
Udbhava sthāna	Āmāśaya
Sañcāra sthāna	Triyakgaḥ sirā
Vyakta sthāna	Tvak
Rogamārga	Bāhya

Pūrvarūpa of Kuṣṭha

- स्पर्शाज्ञत्वमतिस्वेदो न वा वैवर्ण्यमुन्नतिः। कोठानां लोमहर्षश्च कण्डूस्तोदः श्रमः क्लमः॥ व्रणानामधिकं शूलं शीघ्रोत्पत्तिश्चिरस्थितिः। दाहः सुप्ताङ्गता चेति कुष्ठलक्षणमग्रजम्॥ (Ca.Ci.7/11-12)

Sparśājñatva (loss of touch sensation), Atisveda na vā (excessive perspiration or absence of perspiration), Vaivarṇyam (discoloration), Unnati (elevated skin eruptions), Koṭhā (urticarial skin lesions), Lomaharṣa (horripilation), Kaṇḍū (itching sensation), Toda (pricking pain), Śrama (exhaustion), Klama (fatigue), Vraṇāmādhikaṃ śūlaṃ (severe pain in ulcers), Śīghrotpattiścirasthiti (quick appearance and prolonged persistence of these ulcers), Dāha (burning sensation) and Suptāṅgatā (numbness of the body parts) are the premonitory symptoms of Kuṣṭha.

- तस्य पूर्वरूपाणि- त्वक्पारुष्यमकस्माद्रोमहर्षः कण्डूः स्वेदबाहुल्यमस्वेदनं वाऽङ्गप्रदेशानां स्वापः क्षतविसर्पणमसृजः कृष्णता चेति ॥ (Su.Ni.5/4)

Tvakpāruṣya (roughness of the skin), Akasmāt romaharṣa (sudden horripilation), Kaṇḍū (itching sensation), Sveda bāhulyam asvedanaṃ (excessive perspiration or absence of perspiration), Aṅgapradeśānāṃ svāpaḥ (loss of sensation in the body parts), Kṣatavisarpaṇa (spreading of ulcers) and Asṛjaḥ kṛṣṇāta (blackish discoloration of blood) are the premonitory symptoms of Kuṣṭha..

Bheda of Kuṣṭha

- न च किञ्चिदस्ति कुष्ठमेकदोषप्रकोपनिमित्तम्, अस्ति तु खलु समानप्रकृतीनामपि कुष्ठानां दोषांशांशविकल्पानुबन्धस्थानविभागेन वेदनावर्णसंस्थानप्रभावनामचिकित्सितविशेषः। स सप्तविधोऽष्टादशविधोऽपरिसङ्ख्येयविधो वा भवति। दोषा हि विकल्पनैर्विकल्प्यमाना विकल्पयन्ति विकारान्, अन्यत्रासाध्यभावात्। (Ca.Ni.5/4)

No Kuṣṭha manifests due to vitiation of only one Doṣa. Though all the Kuṣṭha have same causative factors, there is much difference in Vedanā (pain), Varṇa (colour), Saṃsthāna (site), Prabhāva (effect), Nāma (name) and Cikitsā (treatment) due to variation in Doṣāṃśāṃśa (degree or stage of doṣas), Anubandha doṣas (their association) and Sthāna (location). Hence, it is said to be of Saptavidha (seven types), Aṣṭadaśavidha (eighteen types) or Aparisaṅkhya (innumerable) types of Kuṣṭha. Depending upon the permutation and combination of Doṣas, variation in types of diseases occurs except in incurable conditions.

- अत ऊर्ध्वमष्टादशानां कुष्ठानां कपालोदुम्बरमण्डलर्ष्यजिह्वपुण्डरीकसिध्मकाकणकैककुष्ठचर्माख्यकिटिमविपादिकालसकदद्रुचर्मदलपामाविस्फोटकशतारुर्विचर्चिकानां ... । (Ca.Ci.7/13)

The eighteen types of Kuṣṭha are Kapāla, Udumbara, Maṇḍala, Ṛṣyajihva, Puṇḍarīka, Sidhma, Kākaṇaka (are seven Mahā Kuṣṭha), Ekakuṣṭha, Carmākhya, Kiṭima, Vipādika, Alasaka, Dadru, Carmadala, Pāma, Visphoṭaka, Śatāru and Vicarcikā (are eleven Kṣudra Kuṣṭha).

Vāgbhaṭa classified the Kuṣṭha similar to Caraka but instead of Sidhma Kuṣṭha he added Dadru Kuṣṭha in Mahā Kuṣṭha and vice-versa.

- ⦿ तत्र सप्त महाकुष्ठानि, एकादश क्षुद्रकुष्ठानि, एवमष्टादश कुष्ठानि भवन्ति । तत्र महाकुष्ठान्यरुणोदुम्बरर्ष्य(र्क्ष)जिह्वकपालकाकणकपुण्डरीकदद्रुकुष्ठानीति । क्षुद्रकुष्ठान्यपि स्थूलारुष्कं महाकुष्ठमेककुष्ठं चर्मदलं विसर्पः परिसर्पः सिध्मं विचर्चिका किटिभं (मं) पामा रकसा चेति ॥ (Su.Ni.5/5)

The seven Mahā Kuṣṭha are Aruṇa, Udumbara, Ṛṣyajihva, Kapāla, Kākaṇaka, Puṇḍarīka and Dadru. The eleven Kṣudra Kuṣṭha are Sthūlāruṣka, Mahākuṣṭham, Ekakuṣṭham, Carmadala, Visarpa, Parisarpa, Sidhma, Vicarcikā, Kiṭibha, Pāmā and Rakasā.

S.No	Kuṣṭha Bheda	Caraka	Suśruta	Vāgbhaṭa
	Mahā Kuṣṭha			
1	Kapāla	+	+	+
2	Udumbara	+	+	+
3	Maṇḍala	+	–	+
4	Ṛṣyajihva	+	+	+
5	Puṇḍarīka,	+	+	+
6	Sidhma	+	–	–
7	Kākaṇaka	+	+	+
8	Dadrū	–	+	+
9	Aruṇa	–	+	–
	Kṣudra Kuṣṭha			
1	Ekakuṣṭha	+	+	+
2	Carmākhya	+	–	+
3	Kiṭima	+	+	+
4	Vipādika	+	–	+
5	Alasaka	+	–	+
6	Dadru	+	–	–
7	Carmadala	+	+	+
8	Pāma	+	+	+
9	Visphoṭāka	+	–	+
10	Śatāru	+	–	+
11	Vicarcikā	+	+	+
12	Sthūlāruṣka	–	+	–
13	Mahākuṣṭha	–	+	–
14	Visarpa	–	+	–
15	Parisarpa	–	+	–
16	Sidhma	–	+	+
17	Rakasā	–	+	–

Doṣa predominance in Kuṣṭha

- ⦿ तत्र वातेनारुणं, पित्तेनोदुम्बरर्ष्य(र्क्ष)जिह्वकपालकाकणकानि, श्लेष्मणा पुण्डरीकं दद्रुकुष्ठं चेति । तेषां महत्त्वं क्रियागुरुत्वमुत्तरोत्तरं धात्वनुप्रवेशादसाध्यत्वं चेति ॥ (Su.Ni.5/7)

Vāta is predominant in Aruna, Pitta in Udumbara, Ṛṣyajihva, Kapāla and Kākaṇakā and Kapha in Puṇḍarīka and Dadru. The significance of this order is because of Kriyāgurutvamuttarottaraṃ (increasing difficulty in their treatment), Dhātvanupraveśat (their ability to involve the dhātus successively) and Asādhyatvaṃ (their increasing incurability).

- ⦿ अरुः ससिध्मं रकसा महच्च यच्चैककुष्ठं कफजान्यमूनि । वायोः प्रकोपात् परिसर्पमेकं शेषाणि पित्तप्रभवाणि विद्यात् ॥ (Su.Ni.5/16)

Sthūlāruṣka, Sidhma, Rakasā, Mahākuṣṭham and Ekakuṣṭham are produced by Kapha doṣa; Parisarpa is produced by Vāta doṣa and Carmadala, Visarpa, Vicarcikā, Kiṭibha, and Pāmā are produced by Pitta doṣa.

- ⦿ वातेऽधिकतरे कुष्ठं कापालं मण्डलं कफे। पित्ते त्वौदुम्बरं विद्यात् काकणं तु त्रदोषजम्॥
  वातपित्ते श्लेष्मपित्ते वातश्लेष्मणि चाधिके। ऋष्यजिह्वं पुण्डरीकं सिध्मकुष्ठं च जायते॥
  चर्माख्यमेककुष्ठं च किटिमं सविपादिकम्। कुष्ठं चालसकं ज्ञेयं प्रायो वातकफाधिकम्॥
  पामा शतारुर्विस्फोटं दद्रुश्चर्मदलं तथा। पित्तश्लेष्माधिकं प्रायः कफप्राया विचर्चिका॥ (Ca.Ci.7/27-30)
- ⦿ वातेन कुष्ठं कापालं, पित्तादौदुम्बरं, कफात्॥ मण्डलाख्यं विचर्ची च, ऋक्षाख्यं वातपित्तजम्।
  चर्मैककुष्ठकिटिभसिध्मालसविपादिकाः॥ वातश्लेष्मोद्भवाः, श्लेष्मपित्ताद्दद्रुशतारुषी।
  पुण्डरीकं सविस्फोटं पामा चर्मदलं तथा॥ सर्वैः स्यात्काकणं.. । (A.H.Ni.14/7-10)

Vāta is predominant in Kapāla, Kapha in Maṇḍala, Pitta in Udumbara, Tridoṣa in Kākanaka, Vātapitta in Ṛṣyajihva, Śleṣmapitta in Puṇḍarīka and Vātaśleṣma in Sidhma mahā kuṣṭha.

In Kṣudra kuṣṭha, Vātakapha doṣas are predominant in Carmākhya, Ekakuṣṭha, Kiṭima, Vipādika and Alasaka; Pittakapha doṣas are predominant in Pāmā, Śatāru, Visphoṭāka, Dadru and Carmadala; and Kapha doṣa predominant in Vicarcikā.

Doṣaja bheda and Lakṣaṇās

- ⦿ रौक्ष्यं शोषस्तोदः शूलं सङ्कोचनं तथाऽऽयामः। पारुष्यं खरभावो हर्षः श्यावारुणत्वं च॥
  कुष्ठेषु वातलिङ्गं, दाहो रागः परिस्रवः पाकः। विस्रो गन्धः क्लेदस्तथाऽङ्गपतनं च पित्तकृतम्॥

श्वैत्यं शैत्यं कण्डूः स्थैर्यं चोत्सेधगौरवस्नेहाः। कुष्ठेषु तु कफलिङ्गं जन्तुभिरभिभक्षणं क्लेदः॥ (Ca.Ci.7/34-36)

- ⊙ कुष्ठेषु तु त्वक्सङ्कोचस्वापस्वेदशोफभेदकौण्यस्वरोपघाता वातेन, पाकावदरणाङ्गुलिपतन कर्णनासाभङ्गाक्षिरागसत्त्वोत्पत्तयः पित्तेन, कण्डूवर्णभेदशोफास्रावगौरवाणि श्लेष्मणा ॥ (Su.Ni.5/18)

The clinical features of Vātaja Kuṣṭha are Raukṣyam (roughness), Śoṣa (wasting), Toda (pricking pain), Śūla (colicky pain), Saṇkoca (contracture), Āyāma (expansion), Pāruṣya (hardness), Kharabhāva (coarseness), Harṣa (horripilation) and Śyāvāruṇa (dark brownish or reddish in colour).

The clinical features of Pittaja Kuṣṭha are Dāha (burning sensation), Rāga (redness), Parisrava (discharges), Pāka (suppuration), Visra gandha (foul smell), Kleda (moistness) and Aṅgapatana (sloughing of body parts).

The clinical features of Kaphaja Kuṣṭha are Śvaityaṃ (whiteness), Śaityaṃ (coldness), Kaṇḍū (itching), Sthairyaṃ (stability), Utsedha (elevation), Gaurava (heaviness), Snehā (unctuousness) Jantubhirabhi bhakṣaṇaṃ (eating away by worms) and Kleda (moistness).

Lakṣaṇās of Mahā Kuṣṭha

Kapāla Kuṣṭha

- ⊙ कृष्णारुणकपालाभं यद्रूक्षं परुषं तनु। कापालं तोदबहुलं तत्कुष्ठं विषमं स्मृतम्॥ (Ca.Ci.7/14)
- ⊙ कृष्णारुणकपालाभं रूक्षं सुप्तं खरं तनु॥ विस्तृतासमपर्यन्तं हृषितैर्लोमभिश्चितम्।
  तोदढ्यमल्पकण्डूकं कापालं शीघ्रसर्पि च। (A.H.Ni.14/13-14)

The clinical features of Kapāla Kuṣṭha are Kṛṣṇāruṇa varṇa (blackish or reddish in colour), resembles Kapāla (broken earthen pot), Rūkṣa (dry), Parūsa (rough), Tanu (thin) associated with Toda bahulaṃ (severe pricking type of pain) and it is Viṣamaṃ (difficult to cure).

Vāgbhaṭa added the features like Supta (numbness), Vistṛtāsamaparyantaṃ (wide with uneven edges), Hṛṣitairlomabhiścita (lesion covered with hairs), Alpa kaṇḍūkaṃ (minimal itching sensation) and Śīgrasarpi (spreads quickly).

- ⊙ कृष्णकपालिकाप्रकाशानि कपालकुष्ठानि, ॥ (Su.Ni.5/8)

Kapāla Kuṣṭha resembles Kṛṣṇakapālikāprakāśāni (black earthen pot).

Aruṇa Kuṣṭha

- ⊙ तत्र, वातेनारुणाभानि तनूनि विसर्पीणि तोदभेदस्वापयुक्तान्यरुणानि । (Su.Ni.5/8)

Aruṇa Kuṣṭha, caused by Vāta doṣa is characterized by Aruṇābha (reddish in colour), Tanūni (thin), Visarpīṇi (spreads quickly), Toda (pricking pain), Bheda (tearing pain) and Svāpayukta (loss of sensation).

Udumbara Kuṣṭha

- ⊙ दाहकण्डूरुजारागपरीतं लोमपिञ्जरम्। उदुम्बरफलाभासं कुष्ठमौदुम्बरं विदुः॥ (Ca.Ci.7/15)
- ⊙ पक्वोदुम्बरताम्रत्वग्रोम गौरसिराचितम्। बहलं बहलक्लेदरक्तं दाहरुजाधिकम्॥
  आशूत्थानावदरणकृमि विद्यादुदुम्बरम्। (A.H.Ni.14/15-16)

The clinical features of Udumbara Kuṣṭha are Dāha (burning sensation), Kaṇḍū (itching sensation), Rujā (pain), Rāgaparīta (redness), Lomapiñjara (brown skin hair) and it resembles Udumbara phala (fruit of Udumbara) and it is predominant of Pitta doṣa.

Vāgbhaṭa added the features like Tāmrātvagroma (coppery skin hairs), Gaurasirācitam (with whitish veins over the lesion), Bahala (thick), Bahalakledarakta (excessive discharge of moistness and blood), Āśūtthāna (rapid onset of skin lesion), Avadaraṇa (rapid onset of putrefaction) and Kṛmi (appearance of worms).

- ⊙ पित्तेन पक्वोदुम्बरफलाकृतिवर्णान्यौदुम्बराणि । (Su.Ni.5/8)

Udumbara Kuṣṭha caused by Pitta doṣa is characterized by Ākṛti (shape and size) and Varṇa (colour) of the lesion similar to that of Udumbara phala (fruit of Udumbara).

Maṇḍala Kuṣṭha

- ⊙ श्वेतं रक्तं स्थिरं स्त्यानं स्निग्धमुत्सन्नमण्डलम्। कृच्छ्रमन्योन्यसंसक्तं कुष्ठं मण्डलमुच्यते॥ (Ca.Ci.7/16)
- ⊙ स्थिरं स्त्यानं गुरु स्निग्धं श्वेतरक्तमनाशुगम्॥ अन्योन्यसक्तमुत्सन्नं बहुकण्डूस्रुतिक्रिमि।
  श्लक्ष्णपीताभपर्यन्तं मण्डलं परिमण्डलम्। (A.H.Ni.14/16-17)

The clinical features of Maṇḍala Kuṣṭha are Śveta rakta varṇa (whitish or reddish in colour); Sthira (stable), Styāna (dense), Snigdha (unctuous), Utsanna maṇḍala (elevated round patches) and Anyonyasaṃsaktaṃ (adhered with other lesions) and it is predominant of Kapha doṣa.

Vāgbhaṭa added the features like Anāśugam (slow progressive), Bahu kaṇḍūśrutikṛmi (excessive itching, discharge and appearance of worms) and Ślakṣṇapītābhaparyantaṃ (yellowish smooth edges)

Ṛṣyajihva Kuṣṭha

- ⊙ कर्कशं रक्तपर्यन्तमन्तः श्यावं सवेदनम्। यदृष्यजिह्वासंस्थानमृष्यजिह्वं तदुच्यते॥ (Ca.Ci.7/17)
- ⊙ परुषं तनु रक्तान्तमन्तःश्यावं समुन्नतम्॥ सतोददाहरुक्क्लेदं कर्कशैः पिटिकैश्चितम्।
  ऋक्षजिह्वाकृति प्रोक्तमृक्षजिह्वं बहुक्रिमि॥ (A.H.Ni.14/18-19)

The clinical features of Ṛṣyajihva Kuṣṭha are Karkaśa (rough), Rakta paryantamantaḥ Śyāvaṃ (the lesion is dark brown in colour in the middle with red colour margins) Savedanam (painful) and it resembles Ṛṣyajihva (tongue of Ṛṣya – Nīlaṇḍa hariṇa (type of deer with blue scrotum) and it is predominant of Vāta and Pitta doṣa.

Vāgbhaṭa added the features like Paruṣa (hard), Tanu (thin), Samunnatam (elevated), Śatodadāharukkledaṃ (with pricking pain, burning sensation, pain and moistness), Piṭikaiścitam (studded with eruptions) and Bahu kṛmi (having excessive worms)

⊙ ऋष्य(क्ष)जिह्वाप्रकाशानि खराणि ऋष्य(क्ष)जिह्वानि । (Su.Ni.5/8)

Ṛṣyajihva Kuṣṭha is Ṛṣyajihva prakāśāni (similar to the tongue of Ṛṣya (type of deer)) associated with Kharāṇi (roughness).

Puṇḍarīka Kuṣṭha

⊙ सश्वेतं रक्तपर्यन्तं पुण्डरीकदलोपमम्। सोत्सेधं च सदाहं च पुण्डरीकं तदुच्यते॥ (Ca.Ci.7/18)

⊙ रक्तान्तमन्तरा पाण्डु कण्डूदाहरुजान्वितम्। सोत्सेधमाचितं रक्तैः पद्मपत्रमिवांशुभिः॥
घनभूरिलसीकासृक्प्रायमाशु विभेदि च। पुण्डरीकम् ... ॥ (A.H.Ni.14/26-27)

The clinical features of Puṇḍarīka Kuṣṭha are Saśvetaṃ rakta paryantaṃ (white lesions with red margins), it resembles Puṇḍarīkadalopamam (lotus petals), Sa utsedhaṃ (elevated) and Sadāha (burning sensation) and it is predominant of Pitta and Kapha doṣa.

Vāgbhaṭa added the features like Kaṇḍū (itching), Rujā (thin), Ācitaṃ raktaiḥ Padmapatrabhivāṃśubhiḥ (covered with reddish dust resembling the lotus petal) and Ghanabhūrilasīkāsṛkprāyamaśu (thick and copious discharge of lymph and blood and spreads quickly)..

⊙ श्लेष्मणा पुण्डरीकपत्रप्रकाशानि पौण्डरीकाणि । (Su.Ni.5/8)

Puṇḍarīka Kuṣṭha is caused by Śleṣma doṣa and it is Puṇḍarīkapatra prakāśāni (resembles the leaves of white lotus).

Sidhma Kuṣṭha

⊙ श्वेतं ताम्रं तनु च यद्रजो घृष्टं विमुञ्चति। अलाबूपुष्पवर्णं तत् सिध्मं प्रायेण चोरसि॥ (Ca.Ci.7/19)

The clinical features of Sidhma Kuṣṭha are Śvetaṃ tāmraṃ (whitish or coppery colour), Tanu (thin), Yadrajo ghṛṣṭaṃ vimuñcati (dust like substances appears while rubbing), it resembles Alābū puṣpavarṇa (colour of Alābu flower) and usually appears on Uras (chest) and it is predominant of Kapha and Vāta doṣa.

Vāgbhaṭa and Suśruta classified Sidhma Kuṣṭha as Kṣudra Kuṣṭha.

⊙ सिध्मं रूक्षं वहिः स्निग्धमन्तर्घृष्टं रजः किरेत्॥ श्लक्ष्णस्पर्शं तनु श्वेतताम्रं दौग्धिकपुष्पवत्। प्रायेण चोर्ध्वकाये स्यात्... । (A.H.Ni.14/21-23)

⊙ कण्ड्वन्वितं श्वेतमपायि सिध्म विद्यात्तनु प्रायश ऊर्ध्वकाये । (Su.Ni.5/12)

Suśruta described the clinical features of Sidhma Kuṣṭha as Kaṇḍū (itching) associated with Śvetaṃ (whitish in colour), Tanu (thin) and Prāyaśa ūrdhvakāye (appears especially in the upper part of the body).

Vāgbhaṭa added Rūkṣa bahiḥ snigdhamantaḥ (non-unctuous outside and unctuous inside), Ghṛṣṭam rajaḥ kiret (dust appears on scratching), Ślakṣṇa sparśa (smooth to touch), Tanu (thin), Śveta tāmraṃ (whitish or coppery in colour) and Daugdhika puṣpavat prāyeṇa ūrdhva kāye (resembles the colour of Alābu flower especially in the upper part of the body).

Kākaṇaka Kuṣṭha

⊙ यत् काकणन्तिकावर्णमपाकं तीव्रवेदनम्। त्रिदोषलिङ्गं तत् कुष्ठं काकणं नैव सिध्यति॥ (Ca.Ci.7/20)

⊙ ....काकणं तीव्रदाहरुक्॥ पूर्वं रक्तं च कृष्णं च काकणन्तीफलोपमम् ॥ (A.H.Ni.14/29-30)

The clinical features of Kākaṇaka Kuṣṭha are Apāka (does not suppurate), Tīvra vedana (severe pain), it resembles Kākaṇātikā Varṇa (colour of Guñja) and it manifests with all the features of Tridoṣa and it is Asādhya (incurable).

Vāgbhaṭa added the features like Tīvra dāha (severe burning sensation) and Pūrvaṃ raktaṃ ca kṛṣṇaṃ ca (reddish and blackish in colour at the time of onset).

⊙ काकणन्तिकाफलसदृशान्यतीव रक्तकृष्णानि काकणकानि । (Su.Ni.5/8)

Kākaṇaka Kuṣṭha is characterized by Kākaṇantikāphala sadṛśānyatīva (resembles the colour of Guñja phala) and Raktakṛṣṇāni (reddish or blackish in colour)

Lakṣaṇās of Kṣudra Kuṣṭha

Ekakuṣṭha

⊙ अस्वेदनं महावास्तु यन्मत्स्यशकलोपमम्। तदेककुष्टं, ...। (Ca.Ci.7/21)

⊙ .... एकाख्यं महाश्रयम्। अस्वेदं मत्स्यशकलसन्निभम् ॥ (A.H.Ni.14/20)

The clinical features of Ekakuṣṭha are Asvedanam (absence of perspiration), Mahāvāstu (covers large area) and Matsyaśakalopamam (resembles fish scales). Vāgbhaṭa stated Mahāśraya (covers large area) instead of Mahāvāstu.

⊙ कृष्णारुणं येन भवेच्छरीरं तदेककुष्ठं प्रवदन्ति कुष्ठम् । (Su.Ni.5/10)

Kṛṣṇāruṇa bhavet śarīra (blackish red discoloration of body) is the clinical features of Ekakuṣṭha.

Carmākhya

⊙ चर्माख्यं बहलं हस्तिचर्मवत्॥ (Ca.Ci.7/21)

⊙ हस्तिचर्मखरस्पर्शं चर्म ... । (A.H.Ni.14/20)

The clinical features of Carmākhya are Bahala (thick skinned) and Hasti carmavat (resembles the skin of elephant). Vāgbhaṭa added Khara sparśa (rough to touch).

Kiṭima or Kiṭibha

⊙ श्यावं किणखरस्पर्शं परुषं किटिमं स्मृतम्। (Ca.Ci.7/22)

⊙ ....किटिभं पुनः॥ रूक्षं किणखरस्पर्शं कण्डूमत्परुषासितम्। (A.H.Ni.14/20-21)

The clinical features of Kiṭima are Śyāva (dark brown coloured), Kiṇakhara sparśa (rough in touch like a scar tissue) and Paruṣam (hard). Vāgbhaṭa added Rūkṣa (non-unctuous / dry skin) and Kaṇḍū (itching)

⊙ यत् स्रावि वृत्तं घनमुग्रकण्डु तत् स्निग्धकृष्णं किटिभं(मं) वदन्ति । (Su.Ni.5/14)

Kiṭibha Kuṣṭha is characterized by Srāvi (skin patches with discharges), Vṛttaṃ (circular skin patches) which are Ghana (dense), Ugrakaṇḍū (has severe itching), Snigdha (slimy) and Kṛṣṇa (blackish in colour).

Vaipādika

⊙ वैपादिकं पाणिपादस्फुटनं तीव्रवेदनम्॥ (Ca.Ci.7/22)

⊙ ...पाणिपाददार्यो विपादिकाः। तीव्रार्त्यो मन्दकण्ड्वश्च सरागपिटिकाचिताः॥ (A.H.Ni.14/23)

The clinical features of Vaipādika are Paṇipādasphuṭanaṃ (cracks on the palms and soles) and Tīvra vedanam (severe pain). Vāgbhaṭa added Manda Kaṇḍū (mild itching) and Sarāga piṭikā (with red coloured eruptions)

⊙ कण्डूमती दाहरुजोपपन्ना विपादिका पादगतेयमेव ॥ (Su.Ni.5/13)

Vaipādika Kuṣṭha is characterized by Kaṇḍūmatī (excessive itching), Dāha (burning sensation), Rujopapannā (pain) and Pādagata (cracks in the feet).

Alasaka

⊙ कण्डूमद्भिः सरागैश्च गण्डैरलसकं चितम्। (Ca.Ci.7/23)

⊙ ..... गण्डैः कण्ड्वयुतैश्चितम्॥ रक्तैरलसकम् ....॥ (A.H.Ni.14/22-23)

The clinical features of Alasaka are Kaṇḍū (itching), Sarāga (redness) and Gaṇḍa (appearance of nodules).

Dadru

⊙ सकण्डूरागपिडकं दद्रुमण्डलमुद्गतम्॥ (Ca.Ci.7/23)

The clinical features of Dadru are Sakaṇḍū (itching), Rāga (redness) and Piḍakaṃ Maṇḍalamudgatam (elevated round patch with eruptions).

Vāgbhaṭa and Suśruta explained Dadru Kuṣṭha in Mahā Kuṣṭha.

⊙ अतसीपुष्पवर्णानि ताम्राणि वा विसर्पीणि पिडकावन्ति च दद्रुकुष्ठानि, ॥ (Su.Ni.5/8)

⊙ दीर्घप्रताना दूर्वावदतसीकुसुमच्छविः। उत्सन्नमण्डला दद्रूः कण्डूमत्यनुषङ्गिणी॥ (A.H.Ni.14/24)

Dadru Kuṣṭha is characterized by Atasīpuṣhpa varṇāni (colour of Atasī flower), Tāmrāṇi (coppery colour), Visarpīṇi Piḍakāvanti (eruptions which spreads quickly).

Vāgbhaṭa added the features like Dīrghapratānā dūrvāvada (skin patches spreads long and wide resembling the Dūrvā grass), Atasīkusumacchaviḥ (has the colour similar to that of Atasī flower), Utsanna maṇḍalā (elevated circular patches) associated with Kaṇḍū (itching) and Atyanuṣaṅghinī (persists for long time).

Carmadala

⊙ रक्तं सकण्डु सस्फोटं सरुग्दलति चापि यत्। तच्चर्मदलमाख्यातं संस्पर्शासहमुच्यते॥ (Ca.Ci.7/24)

⊙ सस्फोटमस्पर्शसहं कण्डूषातोददाहवत्। रक्तं दलच्चर्मदलम् ......। (A.H.Ni.14/29)

The clinical features of Carmadala are Rakta (reddish), Sakaṇḍū (itching), Sasphoṭaṃ (has blisters), Sarugdalati (blisters burst with pain) and Saṃsparśāsaham (tenderness to touch). Vāgbhaṭa added Oṣa (heat), Toda (pricking pain) and Dāhavat (burning sensation).

⊙ स्युर्येन कण्डूव्यथनौषचोषास्तलेषु तच्चर्मदलं वदन्ति ॥ (Su.Ni.5/10)

Carmadala Kuṣṭha is characterized by Kaṇḍū (itching), Vyatha (pain), Oṣa (heat) and Coṣāstaleṣu (burning or sucking sensation in the palms).

Pāmā

⊙ पामाश्वेतारुणश्यावाः कण्डूलाः पिडका भृशम्। (Ca.Ci.7/25)

⊙ ..... पिटिकाः पामा कण्डूक्लेदरुजाधिकाः। सूक्ष्माः श्यावारुणा बह्व्यः प्रायः स्फिक्पणिकूर्परे॥ (A.H.Ni.14/28)

The clinical features of Pāmā are Piḍakā bhṛśam (eruptions) associated with Śvetāruṇaśyāvāḥ (whitish, reddish or dark brownish colour) and Kaṇḍūlāḥ (itching). Vāgbhaṭa added Kledarujādhikā (excessive moistness and pain), Sūkṣmāḥ (small in size), Śyāvāruṇā (dark brownish or reddish skin lesion), Bahvyaḥ (numerous) and Prāyaḥ sphikpaṇikūrpare (appears especially in buttocks, hands and elbows).

⊙ सास्रावकण्डूपरिदाहकाभिः पामाऽणुकाभिः पिडकाभिरूह्या ॥ स्फोटैः सदाहैरति सैव कच्छूः स्फिक्पाणिपादप्रभवैर्निरूप्या। (Su.Ni.5/14-15)

Pāmā Kuṣṭha is characterized by Sasrāva (with discharge), Kaṇḍū (itching), Paridāhakābhiḥ (burning sensation) and Aṇukābhiḥ piḍakābhirūhyā (appearance of minute eruptions). If the Pāmā Kuṣṭha is associated with Sadāha Sphoṭā (blisters with burning sensation) especially appearing in Sphikpāṇipāda (buttocks, hands and feet) it is known as **Kacchū Kuṣṭha.**

Visphoṭāka

⊙ स्फोटाः श्वेतारुणाभासो विस्फोटाः स्युस्तनुत्वचः॥ (Ca.Ci.7/25)

- ⦿ ......तनुत्वग्भिश्चितं स्फोटैः सितारुणैः॥ विस्फोटम्......॥ (A.H.Ni.14/27-28)

The clinical features of Visphoṭāka are Sphoṭā (blisters) associated with Śvetāruṇābhāsā (whitish or reddish colour) and Tanutvacaḥ (thin skin). Vāgbhaṭa added Sphoṭaiḥ Sitāruṇaiḥ (blackish red blisters)

Śatāru

- ⦿ रक्तं श्यावं सदाहार्ति शतारुः स्याद्बहुव्रणम्। (Ca.Ci.7/26)
- ⦿ स्थूलमूलं सदाहार्ति रक्तश्यावं बहुव्रणम्। शतारुः क्लेदजन्त्वाढ्यं प्रायशः पर्वजन्म च॥ (A.H.Ni.14/25)

The clinical features of Śatāru are Raktaṃ śyāvaṃ (reddish or dark brownish), Bahuvraṇam (multiple ulcers) associated with Sadāhārti (burning sensation and pain). Vāgbhaṭa added Sthūlamūla (deep seated), Kledajantvāḍhyaṃ (has excessive moistness and worms) and Prāyaśa parvajanma (appears especially on joints)

Vicarcikā

- ⦿ सकण्डूः पिडका श्यावा वहुस्रावा विचर्चिका॥ (Ca.Ci.7/26)
- ⦿ सकण्डूपिटिका श्यावा लसीकाढ्या विचर्चिका। (A.H.Ni.14/18)

The clinical features of Vicarcikā are Śyāva piḍakā (dark brownish eruptions) associated with Sakaṇḍū (itching) and Bahusrāvā (excessive discharges). Vāgbhaṭa added Lasīkāḍhyā (excessive watery discharge)

- ⦿ राज्योऽतिकण्ड्वर्तिरुजः सरूक्षा भवन्ति गात्रेषु विचर्चिकायाम् । (Su.Ni.5/13)

Vicarcikā Kuṣṭha is characterized by Rājyo (cracks), Atikaṇḍvartirujaḥ (excessive itching and pain) and Sarūkṣā gātresu (roughness in the body)

Sthūlāruṣka

- ⦿ स्थूलानि सन्धिष्वतिदारुणानि स्थूलारुषि स्युः कठिनान्यरूंषि । (Su.Ni.5/9)

Sthūlāruṣka Kuṣṭha is characterized by Kaṭhinānyarumṣi (hard eruptions) occurring especially in Sandhi (joint) and Atidāruṇāni (in a severe form).

Mahākuṣṭha

- ⦿ त्वक्कोचभेदस्वपनाङ्गसादाः कुष्ठे महत्पूर्वयुते भवन्ति ॥ (Su.Ni.5/9)

Mahākuṣṭha is characterized by Tvak kocabhedasvapana (appearance of wrinkles, cracks and numbness in the skin) associated with Aṅga sādāḥ (lethargy).

Visarpa

- ⦿ विसर्पवत् सर्पति सर्वतो यस्त्वग्रक्तमांसान्यभिभूय शीघ्रम् । मूर्च्छाविदाहारतितोदपाकान् कृत्वा विसर्पः स भवेद्विकारः ॥ (Su.Ni.5/11)

Visarpa Kuṣṭha is characterized by Visarpavat sarpati sarvato yastvagrakta māṃsānyabhibūya śīghram (it quickly spreads all over the body by affecting the skin, blood and muscles like erysipelas) and it is associated with Mūrcchā (unconsciousness), Vidāha (burning sensation), Arati (restlessness), Toda (pricking pain) and Pākā (suppuration).

Parisarpa

- ⦿ शनैः शरीरे पिडकाः स्रवन्त्यः सर्पन्ति यास्तं परिसर्पमाहुः । (Su.Ni.5/12)

Parisarpa Kuṣṭha is characterized by Sravntya piḍakā sarpanti śanaiḥ śarīre (discharging eruptions gradually spreading all over the body).

Rakasā

- ⦿ कण्ड्वन्विता या पिडका शरीरे संस्रावहीना रकसोच्यते सा ॥ (Su.Ni.5/15)

Rakasā Kuṣṭha is characterized by Kaṇḍū (itching sensation) associated with Piḍakā saṃsrāvahīnā (blisters without discharge) in the body.

Differentiate Mahā Kuṣṭha and Kṣudra Kuṣṭha

- ⦿ महत्त्वमुत्तरोत्तरधात्वनुप्रवेशात्। एकादशकुष्ठानां क्षुद्रत्वमुत्तरोत्तरधात्वननुप्रवेशात्। (Ḍa.Su.Ni.5/5)
- ⦿ तत्र सप्त महाकुष्ठानीत्यादि। महत्त्वमेषामुत्तरोत्तरधात्वनुप्रवेशात्। धात्वन्तरप्रवेशोऽप्येषां बहुबहुलदोषारम्भात्। अत एव क्रिया महती महाकुष्ठे, क्षुद्रे पुनरल्पा। (Ga.Su.Ni.5/5)
- ⦿ महत्त्वं चैषां शीघ्रमुत्तरोत्तरधात्ववगाहनबहुदोषजत्वक्रियाभूयस्त्वयोगात्॥ (Ma.M.N.49/10-16)
- ⦿ महत्त्वं चैषां शीघ्रमुत्तरोत्तरधात्ववगाहनबहुलदोषजन्यत्वक्रियाभूयस्त्वयोगात्॥ (Āt.M.N.49/10-16)

Mahā Kuṣṭha	Kṣudra Kuṣṭha
Bahudoṣārambhāt	Alpadoṣārambhāt
Uttarotthara dhātvanupraveśāt	Uttarotthara dhātvananupraveśāt
Bahu doṣajanyatva	Alpadoṣajanyatva
Bahu Lakṣaṇa	Alpa Lakṣaṇa
Mahatī kriyā	Alpa kriyā

Dhātugata Kuṣṭha Lakṣaṇās

Tvakgata Kuṣṭha

- ⦿ तत्र त्वचि स्थिते कुष्ठे तोदवैवर्ण्यरूक्षताः॥ (A.H.Ni.14/33)
- ⦿ स्पर्शहानिः स्वेदनत्वमीषत्कण्डूश्च जायते । वैवर्ण्यं रूक्षभावश्च कुष्ठे त्वचि समाश्रिते ॥ (Su.Ni.5/22)

Tvakgata Kuṣṭha is characterized by Toda (pricking pain), Vaivarṇya (discoloration), Rūkṣata (dryness), Sparśahāni (loss of tactile sensation) and Svedanatvamīṣatkaṇḍū (excessive sweating with mild itching sensation).

Raktagata Kuṣṭha

- स्वेदस्वापश्वयथवः शोणिते, । (A.H.Ni.14/34)
- त्वक्स्वापो रोमहर्षश्च स्वेदस्याभिप्रवर्तनम् । कण्डूर्विपूयकश्चैव कुष्ठे शोणितसंश्रिते ॥ (Su.Ni.5/23)

Raktagata Kuṣṭha is characterized by Sveda (excessive sweating), Svāpa (numbness), Śvayathu (swelling), Romaharṣa (horripillation) and Kaṇḍūrvipūyaka (itching and putrid suppuration).

Māṃsagata Kuṣṭha

- पिशिते पुनः। पाणिपादाश्रिताः स्फोटाः क्लेदः सन्धिषु चाधिकम्॥ (A.H.Ni.14/34)
- बाहुल्यं वक्रशोषश्च कार्कश्यं पिडकोद्गमः । तोदः स्फोटः स्थिरत्वं च कुष्ठे मांससमाश्रिते ॥ (Su.Ni.5/24)

Māṃsagata Kuṣṭha is characterized by Pānipādāśritāḥ sphoṭāḥ (blisters appearing in hands and feet), Kleda sandhiṣu cādhikam (moistens excessively especially at joints), Bāhulyaṃ (thick patches) Vaktra śoṣa (drynesss of the mouth), Kārkaśyaṃ (roughness) Piḍakodgama (appearance of eruptions), Toda (with pricking pain), Sphoṭaḥ (blisters) and Sthiratvaṃ (induration).

Medogata Kuṣṭha

- कौण्यं गतिक्षयोऽङ्गानां दलनं स्याच्च मेदसि। (A.H.Ni.14/35)
- दौर्गन्ध्यमुपदेहश्च पूयोऽथ क्रिमयस्तथा । गात्राणां भेदनं चापि कुष्ठे मेदःसमाश्रिते ॥ (Su.Ni.5/25)

Medogata Kuṣṭha is characterized by Kauṇya (falling of body parts such as fingers, toes etc.), Gatikṣaya (restricted movements of the limbs), Aṅgānāṃ dalanaṃ (destruction of body), Daurgandhya (foul smell) Upadeha (slimy coating over the lesion), Pūya (pus formation), Krimayastathā (manifestation of worms) and Gātrāṇāṃ bhedanaṃ (tearing sensation of the body).

Asthimajjāgata Kuṣṭha

- नासाभङ्गोऽस्थिमज्जस्थे नेत्ररागः स्वरक्षयः॥ क्षते च कृमयः, ॥ (A.H.Ni.14/35-36)
- नासाभङ्गोऽक्षिरागश्च क्षते च क्रिमिसम्भवः । भवेत् स्वरोपघातश्च ह्यस्थिमज्जसमाश्रिते ॥ (Su.Ni.5/26)

Asthi and Majjāgata Kuṣṭha are characterized by Nāsābhaṅga (falling of the nose), Netrarogaḥ (diseases of the eyes), Svarakṣaya (loss of voice), Svaropaghāta (impaired speech) and Kṣate ca krimisambhavaḥ (manifestation of worms in the wounds).

Śukragata Kuṣṭha

- शुक्रे स्वदारापत्यबाधनम्। यथापूर्वं च सर्वाणि स्युर्लिङ्गान्यसृगादिषु। (A.H.Ni.14/36)
- कौण्यं गतिक्षयोऽङ्गानां सम्भेदः क्षतसर्पणम् । शुक्रस्थानगते लिङ्गं प्रागुक्तानि तथैव च ॥ (Su.Ni.5/27)

Śukragata Kuṣṭha is characterized by Svadārāpatyabādhanam (afflicts the spouse and progeny of the person), Yathāpūrva ca sarvāṇi liṅgānyasṛgādiṣu (appearance of all the features caused by Raktādi dhātus), Kauṇyaṃ (falling of body parts such as fingers, toes etc.), Gatikṣaya (restricted movements of the limbs), Sambhedaḥ (tearing of the lesions), Kṣatasarpaṇam (spreading of the lesion) and Praguktāni liṅgaṃ (clinical features caused by Raktādi dhātus)

- स्त्रीपुंसयोः कुष्ठदोषाद्दुष्टशोणितशुक्रयोः । यदपत्यं तयोर्जातं ज्ञेयं तदपि कुष्ठितम् ॥ (Su.Ni.5/28)

The child born from a woman and a man with Kuṣṭha doṣāt duṣṭa śoṇita śukrayo (vitiated ovum and sperm due to Kuṣṭha) should be regarded as Kuṣṭhitam.

Sādhyāsādhyata of Kuṣṭha

- सर्वैर्लिङ्गैर्युक्तं मतिमान् विवर्जयेदबलम्। तृष्णादाहपरीतं शान्ताग्निं जन्तुभिर्जग्धम्॥ वातकफप्रबलं यद्यदेकदोषोल्बणं न तत् कृच्छ्रम्। कफपित्त-वातपित्तप्रबलानि तु कृच्छ्रसाध्यानि॥ (Ca.Ci.7/37-38)
- कुष्ठेषु दोषोल्बणताम् सर्वदोषोल्बणं त्यजेत्॥ रिष्टोक्तं यच्च यच्चास्थिमज्जशुक्रसमाश्रयम्। याप्यं मेदोगतम् कृच्छ्रं पित्तद्वन्द्वास्रमांसगम्। अकृच्छ्रं कफवाताढ्यं त्वक्स्थमेकमलं च यत्। (A.H.Ni.14/31-33)

The physician should not be treated the patient of Kuṣṭha having Sarvaliṅga yuktaṃ (all the clinical features) in Abala (weak person), associated with Tṛṣṇā (thirst), Dāha (burning sensation), Śāntāgniṃ (diminished digestive power) and Jantubhirjagdham (presence of worms) as it is incurable.

Kuṣṭha with Vātakapha predominance or Ekadoṣolbaṇaṃ (with one doṣa predominance) is easily curable. Kuṣṭha with Kaphapitta or Vātapitta predominance are Kṛcchra sādhya (difficulty to cure).

Vāgbhaṭa added that the Kuṣṭha located in Asthi, Majjā and Śukra dhātu should be refused; Kuṣṭha located in Medas is Yāpya (palliable); Pittadvandā (Pitta predominance with combination of any other doṣas), localized in Rakta and Māṃsa are Kṛcchra sādhya (difficulty to cure); and Kaphavātāḍhyaṃ (Kapha and Vāta doṣa predominance) localized in Tvak and caused by single doṣa is Na kṛcchra (curable).

- कुष्ठमात्मवतः साध्यं त्वग्रक्तपिशिताश्रितम् । मेदोगतं भवेद्याप्यमसाध्यमत उत्तरम् ॥ (Su.Ni.5/29)

Kuṣṭha located in Tvak, Rakta and Māṃsa are Sādhya (curable); Kuṣṭha located in Medas is Yāpya (palliable); and Asādhya (incurable) if it is located in the rest of the dhātus.

Upadravā of Kuṣṭha

- अस्यां चैवावस्थायामुपद्रवाः कुष्ठिनं स्पृशन्ति; तद्यथा- प्रस्रवणमङ्गभेदः पतनान्यङ्गावयवानां तृष्णाज्वरातीसारदाहदौर्बल्यारोचकाविपाकाश्च, तथाविधमसाध्यं विद्यादिति॥ (Ca.Ni.5/11)

The complications of Kuṣṭha are Prasravaṇa (excessive discharge), Aṅgabhedaḥ (separation of body parts), Patanānyāṅgāvayavānāṃ (falling down of body parts), Tṛṣṇā (thirst), Jvara (fever), Atisāra (diarrhoea), Dāha (burning sensation), Daurbalya (weakness), Arocaka (anorexia), Avipāka (indigestion) and their presence is considered as Asādhya (incurable).

In Contemporary science,

Kuṣṭha can be grossly correlated with Skin disorders right from simple dandruff to severe infective and autoimmune disorders. There are lots of controversies among the Āyurvedic scholars in comparing this concept with modern diseases.

Short Answer Questions

1. Explain Sāmānya Nidāna and Saṃprāpti of Kuṣṭha
2. Explain Dhātugata Kuṣṭha
3. Discuss the classification of Mahā and Kṣudra Kuṣṭha
4. Differentiate Mahā and Kṣudra Kuṣṭha

Long Answer Questions

1. Define Kuṣṭha, and explain the Nidāna, Saṃprāpti, Bheda and Lakṣaṇās of Kuṣṭha
2. Explain Mahā Kuṣṭha in detail
3. Explain Kṣudra Kuṣṭha in detail

◆

TOPIC 30

Śītapitta, Udarda and Koṭha

Introduction

Śītapitta, Udarda and Koṭha are the types of skin disorders. Ācārya Caraka had not mentioned the disease Śītapitta, but he had mentioned Udarda as one among the Kaphaja Nānātmaja Vyādhi and Koṭha as one among the Pittaja Nānātmaja Vyādhi, one of the Santarpaṇotha Vyādhi as well as Rakta pradoṣaja vikāra. Ācārya Suśruta explained Koṭha as one of the symptoms of Mūṣika viṣa (rat bite); whereas Ācārya Vāgbhaṭa mentioned Koṭha under the Kṣudra rogas and as one of the symptoms of improper administration of Vamana karma (emesis). Śītapitta, Udarda and Koṭha are explained together due to the presence of similar features such as Kaṇḍū (itching sensation) and Rāga (redness).

Nidāna and Saṃprāpti of Śītapitta

- ◉ शीतमारुतसंस्पर्शात् प्रदुष्टौ कफमारुतौ । पित्तेन सह सम्भूय बहिरन्तर्विसर्पतः ॥ (M.N.50/1)

Due to exposure to Śītamāruta (cold breeze), the Kapha and Vāta aggravate and vitiate the Pitta, these vitiated doṣas together spreads Bahir (externally to the skin) and Anta (internally to the Raktādi dhātus) and produce the disease Śītapitta.

Saṃprāpti Ghaṭaka of Śītapitta, Udarda and Koṭha

Doṣa	Tridoṣa
Dūṣya	Rasa, Rakta,
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhya janya Āma

Srotas	Rasa, Rakta
Srotoduṣṭi	Saṅga, Vimārga gamana
Adhiṣṭhāna	Tvak
Udbhava sthāna	Āmāśaya
Sañcāra sthāna	Rasa Raktavaha
Vyakta sthāna	Tvak
Rogamārga	Bāhya

Pūrvarūpa of Śītapitta

- पिपासारुचिहृल्लासदेहसादाङ्गगौरवम् । रक्तलोचनता तेषां पूर्वरूपस्य लक्षणम् ॥ (M.N.50/2)

Pipāsā (thirst), Aruci (anorexia), Hṛllāsa (nausea), Dehasāda (malaise), Aṅgagaurava (heaviness of body) and Raktalocana (redness of eyes) are the premonitory symptoms of Śītapitta.

Lakṣaṇās of Śītapitta, Udarda and Koṭha

- वरटीदष्टसंस्थानः शोथः सञ्जायते बहिः । सकण्डूस्तोदबहुलश्छर्दिज्वरविदाहवान् ॥
  उदर्दमिति तं विद्याच्छीतपित्तमथापरे । वाताधिकं शीतपित्तमुदर्दस्तु कफाधिकः ॥ (M.N.50/3-4)

Varaṭidaṃṣṭa saṃsthānaḥ śotha (swelling resembling the sting of wasp) associated with Kaṇḍū bahula (severe itching sensation), Toda bahula (severe pricking pain), Chardi (vomiting), Jvara (fever) and Vidāhavān (burning sensation) are the clinical features of Udarda, also known as Śītapitta by some scholars. However, in Śītapitta, Vāta is predominant whereas in Udarda, Kapha is predominant.

- सोत्सङ्गैश्च सरागैश्च कण्डूमद्भिश्च मण्डलैः । शैशिरः कफजो व्याधिरुदर्द इति कीर्तितः ॥ (M.N.50/5)

Utsaṅga (elevated), Sarāga (reddish), Kaṇḍū (itching sensation) and Maṇḍala (round patches) are the clinical features of Udarda caused by Kapha doṣa in Śiśira Ṛtu (winter season).

- असम्यग्वमनोदीर्णपित्तश्लेष्मान्ननिग्रहैः । मण्डलानि सकण्डूनि रागवन्ति बहूनि च ।
  उत्कोठः सानुबन्धश्च कोठ इत्यभिधीयते ॥ (M.N.50/6)

Asamyak Vamana (improper administration of emesis) and Pittaśleṣmānna Nigraha (obstruction to the movement of Pitta, Kapha and food substances) produces the Maṇḍalāni (round patches) associated with Sakaṇḍūni (itching sensation), Rāgavanti (redness) and Bahūni (numerous in number) is known as Koṭha. If these patches recur again and again, then the condition is known as Utkoṭha.

Differentiate Śītapitta and Udarda

Lakṣaṇās	Śītapitta	Udarda
Doṣa Pradhāna	Vāta Pradhāna	Kapha Pradhāna
Vedanā	Toda	Kaṇḍū
Lakṣaṇa	Varaṭidaṃṣṭa saṃsthānaḥ śotha	Varaṭidaṃṣṭa saṃsthānaḥ śotha or Utsaṅga sarāga kaṇḍū maṇḍala
Anupaśaya	No specific seasonal relation explained for its aggravation	Śiśira sambhava
Svabhāva	Āśukārī	Āśukārī

In Contemporary science,

Śītapitta, Udarda and Koṭha can be correlated with various types of Urticaria (Please refer Topic 33).

Short Answer Questions

1. Explain Śītapitta, Udarda and Koṭha
2. Differentiate Śītapitta and Udarda

Long Answer Questions

1. Explain Śītapitta, Udarda and Koṭha in detail

◆

TOPIC 31

Śvitra

Introduction

Śvitra is one of the Rakta Pradoṣaja Vyādhi and is considered as a type of Kuṣṭha with the difference that Śvitra is Aparisrāvi (without discharge) and Kevalatvaggatavaṃ (involves only skin). Kilāsa, Dāruṇa, Cāruṇa, Pāṇḍura Kuṣṭha, Śveta Kuṣṭha and Varuṇa are the synonyms of Śvitra.

Nirūkti and Paribhāṣa

- श्वेतते इति श्वित्रं । (SKD)
- श्वितत्तं श्वित्रं । (SKD)

That which causes whitish discoloration of the skin is called Śvitra.

- श्वित्रसंज्ञकमिति । श्वेतकुष्ठं अजितवर्णमिति लोके । (Āḍ.Śā.P.7/90)

The skin becomes whitish instead of dark (not faded) complexion in the disease called Śvitra

- श्वेतभावाच्छिवत्रं ॥ (Kā.Sa.Ci.9/2)

Due to the whitish discoloration of the skin, it is called Śvitra

- पित्तञ्चैव गदं भूत्वा वातेनैव समीरितम् । सरक्तञ्च प्रकुपितं कुरुते पाण्डुरच्छविम् ॥ (Hā.Sa.Tṛ.39/50)

The aggravated Vāta vitiates Pitta and afflicts the Rakta dhātu, produces the Pāṇḍura Varṇa (whitish discoloration) of the skin i.e. called as Śvitra.

- त्वग्गतं तु यदस्रावि किलासं तत् प्रकीर्तितम्। यदा त्वचमतिक्रम्य तद्धातूनवगाहते॥ हित्वा किलाससञ्ज्ञां च श्वित्रसञ्ज्ञां लभेत तत्"- इति। (Ḍa.Su.Ni.5/17)

That which surpasses the Tvak (skin) and afflicts the consecutive dhātus such as Rakta, Māṃsa and Medas is termed as Kilāsa or Śvitra.

- त्वग्गतमेव किलासमपरिस्रावि च । (Su.Ni.5/17)

The disease which involves only Tvak dhātu (skin) and is characterized by Aparisrāva (non exudative skin lesion) is called Kilāsa.

- कुष्ठैकसम्भवं श्वित्रं किलासं दारुणं च तत्। निर्दिष्टमपरिस्रावि त्रिधातूद्भवसंश्रयम्॥ (A.H.Ni.14/37)

Śvitra, also known as Kilāsa and Dāruṇa, which is similar to the Kuṣṭha in causative factors, is characterized by Aparisrāvi (non discharging skin lesion) and arises from Tridoṣa (Vāta, Pitta and Kapha) and Tridhātu (Rakta, Māṃsa and Medas).

Nidāna of Śvitra

- वचांस्यतथ्यानि कृतघ्नभावो निन्दा सुराणां गुरुधर्षणं च। पापक्रिया पूर्वकृतं च कर्म हेतुः किलासस्य विरोधि चान्नम्॥ (Ca.Ci.7/177)

Vacāṃsyatathyāni (untruth statements), Kṛtaghnabhavo (ingratitude), Nindā surāṇāṃ (disrespect of gods), Gurugharṣaṇaṃ (disrespect of preceptors), Pāpakriyā (sinful acts of present life), Pūrvakṛtaṃ cakarma (misdeeds of previous life) and Virodhi cānna (incompatible food) are the causative factors of Kilāsa.

Samprāpti of Śvitra

- पित्तञ्चैव गदं भूत्वा वातेनैव समीरितम् । सरक्तञ्च प्रकुपितं कुरुते पाण्डुरच्छविम् ॥
  स्तब्धचित्तं विरूपञ्च तस्य च लक्षणम् ॥ (Hā.Sa.Tṛ.39/50-51)

The aggravated Vāta vitiates Pitta and afflicts the Rakta dhātu, leading to the Pāṇḍura Varṇa (whitish discoloration) of the skin i.e. called as Śvitra and it is characterized by Stabdha citta (stiff minded) and Virūpa (deformed or discoloration).

Samprāpti Ghaṭaka

Doṣa	Pitta Pradhāna Tridoṣa
Dūṣya	Rakta, Māṃsa and Medas
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhya janya Āma
Srotas	Rakta, Māṃsa and Medo vaha
Srotoduṣṭi	Saṅga
Adhiṣṭhāna	Tvak

Udbhava sthāna	Āmāśaya
Sañcāra sthāna	Rasāyanis
Vyakta sthāna	Tvak
Rogamārga	Bāhya

Bheda and Lakṣaṇās of Śvitra

Doṣaja bheda and Lakṣaṇās

Śvitra is classified into three types based on the predominance of doṣas; they are Vātaja, Pittaja and Kaphaja

- वातादृूक्षारुणं, पित्तात्ताम्रं कमलपत्रवत्। सदाहं रोमविध्वंसि, कफाच्छ्वेतं घनं गुरु॥
  सकण्डु च, क्रमाद्रक्तमांसमेदःसु चादिशेत्। वर्णेनैवेदृगुभयं कृच्छ्रं तच्चोत्तरोत्तरम्॥ (A.H.Ni.14/38-39)
- किलासमपि कुष्ठविकल्प एव; तत्त्रिविधं- वातेन, पित्तेन, श्लेष्मणा चेति । कुष्ठकिलासयोरन्तरं- त्वग्गतमेव किलासमपरिस्रावि च । तद्वातेन मण्डलमरुणं परुषं परिध्वंसि च, पित्तेन पद्मपत्रप्रतीकाशं सपरिदाहं च, श्लेष्मणा श्वेतं स्निग्धं बहलं कण्डूमच्च । (Su.Ni.5/17)
  - **Vātaja Śvitra** afflicts the Rakta dhātu and is Rūkṣa (dryness), Maṇḍala (circular patches), Paruṣa (indurated), Paridhvaṃsi (dusty particles appears on rubbing) and Aruṇa varṇa (reddish in colour) in the skin and it is Kṛcchra sādhya (difficult to cure)
  - **Pittaja Śvitra** afflicts the Māṃsa dhātu and is Tāmra varṇa (coppery colour), resembles Padma patravat / Kamala patravat (lotus petal); Paridāha / Sadāha (has burning sensation) and Romavidhvaṃsi (loss of hairs in the body) and it is Kṛcchra tara (more difficult to cure)
  - **Kaphaja Śvitra** afflicts the Medodhātu and is Śveta (whitish), Bahala (extensive), Ghana (thick), Guru (heavy) associated with Kaṇḍū (itching sensation) and it is Kṛcchra tama (most difficult to cure).

Nidānokta bheda

- तथा च भोजः- 'श्वित्रं तु द्विविधं प्रोक्तं दोषजं व्रणजं तथा। तत्र मिथ्योपचाराद्धि व्रणस्य व्रणजं स्मृतम्॥ दोषजं च द्विधा प्रोक्तमात्मजं परजं तथा। परगात्रासनस्पर्शाद्यत्तत् परजमुच्यते॥ तदात्मजं विजानीयाद्यद्देहेष्वनिलादिजम्। (Ga.Su.Ni.5/17)

Ācārya Bhoja classified Śvitra into two types based on the causative factors; they are Vraṇaja and Doṣaja Śvitra

Vraṇaja Śvitra is caused by the Mithyopacāra (improper treatment) of Vraṇa. Doṣaja Śvitra

again classified into two types namely Ātmaja and Paraja. Ātmaja is caused by the vitiation of the doṣas in the body; whereas Paraja is caused by Paragātra sparśa (contact with the inflicted body).

Dhātugata bheda and Lakṣaṇās

- दारुणं चारुणं श्वित्रं किलासं नामभिस्त्रिभिः। विज्ञेयं त्रिविधं तच्च त्रिदोषं प्रायशश्च तत्॥
  दोषे रक्ताश्रिते रक्तं ताम्रं मांससमाश्रिते। श्वेतं मेदःश्रिते श्वित्रं गुरु तच्चोत्तरोत्तरम्॥ (Ca.Ci.7/173-174)

Dāruṇa, Cāruṇa and Śvitra are the various synonyms of Kilāsa. All these three types are usually Tridoṣaja.

- **Dāruṇa** – the doṣas are located in **Rakta dhātu**; it is Rakta varṇa (red colour) and Kaṣṭa Sādhya (difficult to cure).
- **Cāruṇa** – the doṣas are located in **Māṃsa dhātu**; it is Tāmra varṇa (coppery colour) and is Kaṣṭa tara Sādhya (more difficult to cure)
- **Śvitra** –the doṣas are located in **Medo dhātu**, it is Śveta varṇa (whitish colour) and is Kaṣṭa tama Sādhya (most difficult to cure).

Sādhyāsādhyata

- यत् परस्परतोऽभिन्नं बहु यद्रक्तलोमवत्। यच्च वर्षगणोत्पन्नं तच्छ्वित्रं नैव सिध्यति॥
  अरक्तलोम तनु यत् पाण्डु नातिचिरोत्थितम्। मध्यावकाशे चोच्छूनं श्वित्रं तत्साध्यमुच्यते॥ (Ca.Ci.7/175-176)

If Śvitra is Parasparatoabhinnaṃ (conjoined together), Bahu (numerous in number), Raktalomavat (red hairs) and Varṣaganotpannaṃ (more than one year old), then it is Asādhya (incurable); If Śvitra is Araktaloma (hairs not in red colour), Tanu (thin patches), Pāṇḍu (pale in colour), Nāticirotthitam (not a chronic one), Madhyāvakaśo (has the space between two patches) and Śūna (slightly swollen), then considered as Sādhya (curable).

- तेषु सम्बद्धमण्डलमन्तेजातं रक्तरोम चासाध्यमग्निदग्धं च ॥ (Su.Ni.5/17)

If Śvitra has Sambaddha Maṇḍala (contiguous circular patches), Antejātaṃ (has situated in end parts such as lips, hand, feet and genitals) which has Rakta roma (red hairs) and Agnidagdha (those develop after burns) are considered as Asādhya (incurable).

- अशुक्लरोमाबहलमसंसृष्टं मिथो नवम्। अनग्निदग्धजं साध्यं श्वित्रं, वर्ज्यमतोऽन्यथा॥
  गुह्यपाणितलौष्ठेषु जातमप्यचिरन्तनम्। (A.H.Ni.14/40-41)

If Śvitra has Aśukla roma (black hairs), Abahala (patches not thick), Asaṃsṛtaṃ (not joined together), Nava (recent onset) and Anagnidagdhajaṃ (not developed after burns) are Sādhya (curable); whereas Varjyomato anyathā (other than above-mentioned types are rejectable); Śvitra situated in Guhyapāṇitāloṣṭa (genitals, hand, feet and lips) in the acute state also should be rejected.

In Contemporary science,

Śvitra can be correlated with Vitiligo (Please refer Topic 33)

Short Answer Questions

1. Explain Sāmānya Nidāna and Saṃprāpti of Śvitra
2. Explain the types of Śvitra
3. Describe Lakṣaṇa and Sādhyāsādhyata of Śvitra`

Long Answer Questions

1. Define Śvitra, and explain the Nidāna, Saṃprāpti, Bheda and Lakṣaṇās of Śvitra.

◆

TOPIC 32

Visarpa

Introduction

Visarpa is one of the Rakta Pradoṣaja Vyādhi and is one of the Upadrava of Chardi vegapratighātat (suppression of urge of vomiting) hence Ācārya Caraka explained it after Chardi Cikitsā. Visarpa is caused by the involvement of seven factors viz., Tridoṣa, Tvak, Rakta, Māṃsa and Lasikā which is similar to Kuṣṭha. However, the difference within both the diseases is that Visarpa is characterized by the spreading nature of doṣas; while Kuṣṭha is manifested with the chronic accumulation of doṣas. Visarpa is considered as a dreadful disease because it occurs in acute form and spreads quickly.

Nirūkti and Paribhāṣa

The term Visarpa has two words i.e. 'Vi' and 'Sarpa'. The word 'Vi' denotes 'Vividha' which means various or different and the word 'Sarpa' denotes 'Sarpaṇa' which means creeping or spreading. Hence the term Visarpa literally means spreading in different or various directions.

- विसर्पणाद्विसर्पः॥ (Āḍ.Śā.P.7/100)

  That disease which spreads in different directions is called Visarpa
- विविधं सर्पति यतो विसर्पस्तेन स स्मृतः। परिसर्पोऽथवा नाम्ना सर्वतः परिसर्पणात्॥ (Ca.Ci.21/11)

The disease which spreads in different directions such as Adha (downwards), Ūrdhva (upwards) and Tīryak (oblique) is called Visarpa. And it is also known as Parisarpa (the word 'Pari' means 'Sarvato' which means all around) because it spreads all over the body.

Sāmānya Nidāna and Saṃprāpti of Visarpa

- लवणाम्लकटूष्णानां रसानामतिसेवनात्। दध्यम्लमस्तुशुक्तानां सुरासौवीरकस्य च॥
  व्यापन्नबहुमद्योष्णरागषाडवसेवनात्। शाकानां हरितानां च सेवनाच्च विदाहिनाम्॥

कूर्चिकानां किलाटानां सेवनान्मन्दकस्य च। दध्नः शाण्डाकिपूर्वाणामासुतानां च सेवनात्॥
तिलमाषकुलत्थानां तैलानां पैष्टिकस्य च। ग्राम्यानूपौदकानां च मांसानां लशुनस्य च॥
प्रक्लिन्नानामसात्म्यानां विरुद्धानां च सेवनात्। अत्यादानाद्दिवास्वप्नादजीर्णाध्यशनात् क्षतात्॥
क्षतबन्धप्रपतनाद्धर्मकर्मातिसेवनात् । विषवाताग्निदोषाच्च विसर्पाणां समुद्भवः॥
एतैर्निदानैर्व्यामिश्रैः कुपिता मारुतादयः। दूष्यान् सन्दूष्य रक्तादीन् विसर्पन्त्यहिताशिनाम्॥ (Ca.Ci.21/16-22)

The causative factors of Visarpa as follows

- Lavaṇāmlakaṭuṣṇa rasānāmatisevanāt (excessive indulgence in salt, sour, pungent and hot substances);
- Intake of Dadhyamla (sour curd), Mastu (curd water), Śuktā (vinegar), Surā (type of alcohol) and Sauviraka (fermented drink);
- Vyāpannabahumadya (excessive quantity of contaminated alcohol),
- Uṣṇarāgaṣāḍava sevanāt (intake of hot condiments and confectionery);
- Śāka sevanat (intake of vegetables),
- Haritānāṃ sevanāt (intake of uncooked substances such as ginger),
- Vidāhi (causes indigestion with burning sensation);
- Use of Kūrcikā (inspissated milk), Kilāṭā (coagulated milk), Mandaka (uncurdled curd), Śāṇḍāki (fermented drink), Tilamāṣakulatthānāṃ tailānāṃ (oils of sesame, black gram, horse gram), Paiṣṭika (paste of pulses), Grāmyānūpaudakānāṃ ca māṃsānāṃ (meat of domestic, marshy and aquatic animals), Laśuna (garlic);
- Intake of Praklinna (putrefied food), Asātmya (unwholesome food) and Viruddhā (incompatible food substances);
- Atyādāna (over eating),
- Divāsvapna (day sleep),
- Ajīrṇāśana (eating during digestion or eating improperly cooked food),
- Adhyāśana (eating before digestion of previous food),
- Kṣatāt (injury),
- Kṣatabandhaprapatanāt (displacement of bandage of wounds),
- Dharma karmāti sevanāt (excessive work under the sun),
- Viṣavātāgni doṣāt (exposure to poisonous air and fire)

Due to the above described causative factors, the aggravated Vātādi doṣas afflicts the Rakta, Māṃsa, Lasikā and Tvak and produces the disease Visarpa in those who intake unwholesome diet.

- त्वङ्मांसशोणितगताः कुपितास्तु दोषाः सर्वाङ्गसारिणमिहास्थितमात्मलिङ्गम् ।
  कुर्वन्ति विस्तृतमनुन्नतमाशु शोफं तं सर्वतो विसरणाच्च विसर्पमाहुः ॥ (Su.Ni.10/3)

The doṣas located in the skin, muscle and blood get aggravated, spreads all over the body without getting localized and produces their own cardinal symptoms such as Vistṛtamanunnatamāśu Śopha (widespread and un-elevated swelling which spreads quickly all over the body) is known as Visarpa.

- ....तत्र दोषा यथायथम्। प्रकोपणैः प्रकुपिता विशेषेण विदाहिभिः॥
  देहे शीघ्रं विसर्पन्ति तेऽन्तरन्तःस्थिता बहिः। बहिःस्था द्वितये द्विस्थाः.... ॥ (A.H.Ni.13/44-45)

Ācārya Vāgbhaṭa stated that in Visarpa the aggravated doṣas (by their respective cause especially Vidāhi āhāra) spread quickly to all the external and internal parts of the body by getting localized into Antaḥsthitā (internally), Bahiḥsthitā (externally) and Ubhayāśrita (both internal and external).

Samprāpti Ghaṭaka

Doṣa	Pitta Pradhāna Tridoṣa
Dūṣya	Tvak, Rakta, Māṃsa, Lasikā
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhya janya Āma
Srotas	Rasa Rakta and Māṃsavaha
Srotoduṣṭi	Saṅga
Adhiṣṭhāna	Tvak; Bāhyābhyantara
Udbhava sthāna	Āmāśaya
Sañcāra sthāna	Raktavaha
Vyakta sthāna	Tvak
Rogamārga	Bāhya and Abhyantara

Visarpa Bheda and Lakṣaṇās

Āśraya Bheda

Visarpa is classified into three types based on their locations namely Bāhya (externally), Abhyantara (internally) and Ubayāśrita (both external and internal).

- मर्मोपघातात् सम्मोहादयनानां विघट्टनात्। तृष्णातियोगाद्वेगानां विषमाणां प्रवर्तनात्॥ विद्याद्विसर्पमन्तर्जमाशु चाग्निबलक्षयात्। अतो विपर्ययाद्बाह्यमन्यैर्विद्यात् स्वलक्षणैः॥ (Ca.Ci.21/26-27)
  - **Antarāśraya Visarpa** : Marmopaghāta (affliction of vital organs), Sammohā (delusion), Ayanānāṃ vighaṭṭanāt (obstruction to the channels of circulation), Tṛṣṇātiyogāt (excessive thirst), Vegānāṃ viṣamāṇāṃ pravartanāt (irregular manifestation of natural urges) and Agnibalakṣaya (diminution of strength of digestive fire) are the clinical features of internal Visarpa.
  - **Bahirāśraya Visarpa** : It is characterized by the clinical features contrary to the internal Visarpa and it also has its own specific features.

Doṣaja bheda

Visarpa is classified into seven types based on the doṣas predominance namely Vātaja, **Pittaja, Kaphaja,** Vātapittaja (Agneya), Pittakaphaja (Kardama), Kaphavātaja (Granthi) and **Sannipātaja. Ācārya** Vāgbhaṭa added Kṣataja Visarpa.

Ācārya Suśruta classified Visarpa into five types namely Vātaja, Pittaja, Kaphaja, **Sannipātaja and Vraṇaja.**

Doṣaja Bheda	Caraka	Suśruta	Vāgbhaṭa	Mādhavakara
Vātaja	+	+	+	+
Pittaja	+	+	+	+
Kaphaja	+	+	+	+
Vātapittaja (Agneya)	+	–	+	+
Pittakaphaja (Kardama)	+	–	+	+
Kaphavātaja (Granthi)	+	–	+	+
Sannipātaja	+	+	+	+
Kṣataja/Vraṇaja	–	+	+	–

Vātaja Visarpa

- रूक्षोष्णैः केवलो वायुः पूरणैर्वा समावृतः। प्रदुष्टो दूषयन् दूष्यान् विसर्पति यथाबलम्॥ (Ca.Ci.21/29)

The Vāta aggravated alone by Rūkṣa (non-unctuous) and Uṣṇa (hot substances) or aggravated by Mārgāvaraṇa, vitiates the dūṣyas and spreads according to their strength.

- तत्र वातात्परीसर्पो वातज्वरसमव्यथः॥ शोफस्फुरणनिस्तोदभेदायामार्तिहर्षवान्। (A.H.Ni.13/47-48)
- वातात्मकोऽसितमृदुः परुषोऽङ्गमर्दसम्भेदतोदपवनज्वरलिङ्गयुक्तः । (Su.Ni.10/4)

The clinical features of Vātaja Visarpa are Vātajajvara samavyatha (symptoms similar to Vātaja Jvara), Śopha (swelling), Sphuraṇa (throbbing), Nistoda (pricking sensation), Bheda (cutting type of pain), Āyāma (stretching), Arti (pain) and Harṣa (horripilation); Suśruta added features such as Asita (black), Mṛdu (soft), Paruṣā (rough) and Aṅgamarda (body pain).

Pittaja Visarpa

- पित्तमुष्णोपचारेण विदाह्यम्लाशनैश्चितम्। दूष्यान् सन्दूष्य धमनीः पूरयन् वै विसर्पति॥ (Ca.Ci.21/31)

Due to intake of Vidāhi (causes indigestion with burning sensation), Amlāśana (eating sour substances) and involving in Uṣṇa upacāra (hot regimens), the Pitta doṣa gets aggravated and vitiates the dūṣyas, enters the Dhamanī and produces the Pittaja Visarpa.

- पित्ताद्द्रुतगतिः पित्तज्वरलिङ्गोऽतिलोहितः। (A.H.Ni.13/48)
- पित्तात्मको द्रुतगतिर्ज्वरदाहपाकस्फोटप्रभेदबहुलः क्षतजप्रकाशः । (Su.Ni.10/5)

The clinical features of Pittaja Visarpa are Drutagatiḥ (spreads quickly), Pittajvaraliṅga (symptoms similar to Pittaja Jvara) and Atilohita (excessive reddish in colour); Suśruta added Dāha (burning sensation), Pāka (suppuration), Sphoṭā prabeda bahula (blisters bounded or fastened together) and Kṣataja prakāśaḥ (reddish like blood)

Kaphaja Visarpa

- स्वाद्वम्ललवणस्निग्धगुर्वन्नस्वप्नसञ्चितः। कफः सन्दूषयन् दूष्यान् कृच्छ्रमङ्गे विसर्पति॥ (Ca.Ci.21/33)

Due to intake of Svādamlalavaṇa snigdhagurvanna (sweet, sour, salt, unctuous and heavy food substances) and indulging in Svapna (excessive sleep), the Kapha doṣa gets aggravated and vitiates the dūṣyas and spreads with difficulty.

- कफात्कण्डूयुतः स्निग्धः कफज्वरसमानरुक्। (A.H.Ni.13/49)
- श्लेष्मात्मकः सरति मन्दमशीघ्रपाकः स्निग्धः सितश्वयथुरल्परुगुग्रकण्डुः । (Su.Ni.10/6)

The clinical features of Kaphaja Visarpa are Kaṇḍūyutaḥ / Ugrakaṇḍū (excessive itching sensation), Snigdha (unctuous) and Kaphajvarasamānaruk (symptoms similar to Kaphaja Jvara); Suśruta added Manda sarati (spreads slowly), Mandaśighrapāka (suppurates slowly), Sita (whitish in colour), Śvayathu (swelling) and Alparuk (mild pain).

Differentiate Doṣaja Visarpa

Lakṣaṇās	Vātaja Visarpa	Pittaja Visarpa	Kaphaja Visarpa
Jvara Lakṣaṇa	Vātaja jvara samavyatha	Pitta jvara liṅga	Kaphajvarasamānaruk or Śīta Jvara
Anubandha Lakṣaṇa	Śopha, Sphuraṇa, Nistoda, Bheda, Āyāma, Arti, Harṣa, Bhrama, Davathu	Dāha, Tṛṣṇā, Mūrcchā, Atimātra sveda, Asvapna, Atilohita	Ugrakaṇḍū, Snigdha, Gaurava, Nidra, Arocaka, Śvayathu
Śphoṭa Lakṣaṇa	Asita, Mṛdu, Paruṣa, Śyāvāruṇabhāsa śvayathu, Śighrabheda	Drutagatiḥ, Pāka, Sphoṭa prabheda bahula, Kṣataja prakāśaḥ	Sita, Alparuk, Manda sarati, Mandaśīghrapāka / Kṛcchrapākaiścirakāri
Srāva Lakṣaṇa	Śyāvairvā tanuviśadāruṇālpāsrāvaiḥ	Haritahāridranīla kṛṣnaraktānāṃ Varṇa srāva	Śvetaṃ picchilaṃ tantumaddham anubaddhaṃ snigdhamāsrāvaṃ sravati
Anya Lakṣaṇa	Aṅgeṣu Pipīlikāsañcāra, Vibaddha vāta mūtra purīṣa	Haritahāridra Netra mūtra varcastvaṃ	Śveta nayana vadana tvaṅmūtra varcastvaṃ

Vātapittaja /Agni Visarpa

◉ वातपित्तं प्रकुपितमतिमात्रं स्वहेतुभिः। परस्परं लब्धबलं दहद्गात्रं विसर्पति॥ (Ca.Ci.21/35)

Vāta and Pitta aggravated excessively due to their respective causative factors and obtaining strength mutually spreads in the body producing Dahadgātraṃ (burning sensation in the body).

◉ वातपित्ताज्ज्वरच्छर्दिमूर्च्छातीसारतृड्भ्रमैः॥ अस्थिभेदाग्निसदनतमकारोचकैर्युतः।
करोति सर्वमङ्गं च दीप्ताङ्गारावकीर्णवत्॥ यं यं देशं विसर्पश्च विसर्पति भवेत्स सः।
शान्ताङ्गारासितो नीलो रक्तो वाऽऽशु च चीयते॥ अग्निदग्ध इव स्फोटैः शीघ्रगत्वाद् द्रुतं च सः।
मर्मानुसारी वीसर्पः स्याद्वातोऽतिवलस्ततः॥ व्यथेताङ्गं हरेत्संज्ञां निद्रां च श्वासमीरयेत्।
हिध्मां च स गतोऽवस्थामीदृशीं लभते न ना॥ क्वचिच्छर्मारतिग्रस्तो भूमिशय्यासनादिषु।
चेष्टमानस्ततः क्लिष्टो मनोदेहश्रमोद्भवाम्॥ दुष्प्रबोधोऽश्नुते निद्रां सोऽग्निवीसर्प उच्यते। (A.H.Ni.13/50-56)

The clinical features of Vātapittaja or Agni Visarpa are Jvara (fever), Chardi (vomiting), Mūrcchā (fainting), Atisāra (diarrhoea), Tṛd (thirst), Bhrama (giddiness), Asthibheda (splitting pain in the bones), Agnisāda (diminished digestive fire), Tamaka (black out), Arocaka (anorexia), Sarvamaṅgaṃ ca dīptāṅgarāvakīrṇavat (feels as if body is covered with burning charcoal), Deśaṃ visarpaśca visarpati bhavetsa saḥ śāntāṅgarāsito nīlo rakto vāaśu ca cīyate (all the parts where it spreads become black, blue or reddish colour like an xtinguished charcoal), Agnidagdha iva sphoṭaiḥ (blisters resembling as though burnt by ire), Śīghragatvād drutaṃ (spreads quickly), Marmānusārī vīsarpa syadvātoatabalastitaḥ (it

pervades vital organs quickly due to the powerful Vāta), Vyathetāṅga (severe pain in the body), Haretsamjñāṃ (loss of consciousness), Nidrā (sleep), Śvāsamīrayet (dyspnoea), Hidhmā (hiccup); Gateavasthāmiddaśī labhate na nā kvacicharmāratigrasto bhūmiśayyāsanādiṣu (the person who has reached this stage, does not find comfort by lying or sitting on the ground or the couch), Ceṣṭamānastataḥ kliṣto manodehaśramodbhavāṃ (makes difficult movements due to exertion of mind and body) and Duṣprabhodoaśnute nidrāṃ (gets sleep from which he cannot be aroused similar to death).

Vātakaphaja / Granthi Visarpa

◉ स्थिरगुरुकठिनमधुरशीतस्निग्धान्नपानाभिष्यन्दिसेविनामव्यायामादिसेविनामप्रतिकर्मशीलानां श्लेष्मा वायुश्च प्रकोपमापद्यते, तावुभौ दुष्टप्रवृद्धावतिबलौ प्रदूष्य दूष्यान् विसर्पाय कल्पेते; (Ca.Ci.21/39)

Due to the indulgence of diet and drinks which are Sthira (firm), Guru (heavy), Kaṭhina (hard), Madhura (sweet), Śīta (cold), Snigdha (unctuous) and Abhiṣyandi (blocks the channels), Avyāyāmādi sevina (absence of physical exercise etc.), Apratikarmaśilānāṃ (absence of purificatory therapies at appropriate seasons), the Kapha and Vāta aggravates excessively vitiates the dūṣyas and produce the disease Granthi Visarpa.

◉ कफेन रुद्धः पवनो भित्त्वा तं बहुधा कफम्॥ रक्तं वा वृद्धरक्तस्य त्वक्सिरास्नावमांसगम्।
दूषयित्वा च दीर्घाणुवृत्तस्थूलखरात्मनाम्॥ ग्रन्थीनां कुरुते मालां रक्तानां तीव्ररुग्ज्वराम्।
श्वासकासातिसारास्यशोषहिध्मावमिभ्रमैः॥ मोहवैवर्ण्यमूर्छाङ्गभङ्गाग्निसदनैर्युताम्।
इत्ययं ग्रन्थिवीसर्पः कफमारुतकोपजः॥ (A.H.Ni.13/56-59)

The Vāta getting obstructed by Kapha, disintegrates Kapha into many parts or vitiates the Rakta situated in the Tvak (skin), Sirā (veins), Snāyu (tendons) and Māṃsa (muscles) in the persons with aggravated Rakta and produces Dīrghāṇuvṛttasthūlakharātmanām Granthīnāṃ mālāṃ raktānāṃ (chain of reddish tumours which are either long, small, round, thick or rough), associated with Tīvraruja (severe pain), Jvara (fever), Śvāsa (dyspnoea), Kāsa (cough), Atisāra (diarrhoea), Āsya śoṣa (dryness of mouth), Hidhmā (hiccup), Vami (vomiting), Bhrama (giddiness), Moha (delusion), Vaivarṇya (discoloration), Mūrcchā (fainting), Aṅgabhaṅga (body pain) and Agnisadana (diminished digestive fire).

Kaphapittaja / Kardama Visarpa

◉ कफपित्तं प्रकुपित्तं बलवत् स्वेन हेतुना। विसर्पत्येकदेशे तु प्रक्लेदयति देहिनम्॥ (Ca.Ci.21/37)

Kapha and Pitta aggravated excessively due to their respective causative factors spreads in the body causing Ekadeśa tu prakledayati dehinam (moistness in the affected part of the body).

◉ कफपित्ताज्ज्वरः स्तम्भो निद्रातन्द्राशिरोरुजः। अङ्गावसादविक्षेपप्रलापारोचकभ्रमाः॥
मूर्च्छाग्निहानिर्भेदोऽस्थ्नां पिपासेन्द्रियगौरवम्। आमोपवेशनं लेपः स्रोतसां स च सर्पति॥
प्रायेणामाशये गृह्णन्नेकदेशं न चातिरुक्। पिटकैरवकीर्णोऽतिपीतलोहितपाण्डुरैः॥

मेचकाभोऽसितः स्निग्धो मलिनः शोफवान् गुरुः। गम्भीरपाकः प्राज्योष्मा स्पृष्टः क्लिन्नोऽवदीर्यते॥
पङ्कवच्छीर्णमांसश्च स्पष्टस्नायुसिरागणः। शवगन्धिश्च वीसर्पं कर्दमाख्यमुशन्ति तम्॥ (A.H.Ni.13/60-64)

The clinical features of Kaphapittaja or Kardama Visarpa are Jvara (fever), Stambha (stiffness), Nidrā (sleep), Tandrā (drowsiness), Śiroruja (headache), Aṅgāvasāda (debility of the body), Vikṣepa (tremors), Pralāpa (delusion), Arocaka (anorexia), Bhrama (giddiness), Mūrcchā (fainting), Agnihāni (diminished digestive fire), Asthibheda (splitting pain in the bones), Pipāsā (thirst), Indriyagaurava (heaviness of the sense organs), Āmopaveśana (evacuation of faeces containing Āma), Srotolepa (coating of channels), Sarpati prāyeṇāmāśaye (usually it starts spreading from Āmāśaya to the other parts), Ekadeśaṃ na cātiruk piṭakairakīrṇoatipītalohitapāṇḍuraiḥ (the eruption are localized to one part and are not very painful, yellowish, reddish or whitish in colour) and it resembles Mecakābhoasita (peacocks feather in colour or blackish), associated with Snigdha (unctuous), Malinaḥ (dirty), Śopha (swelling), Guru (heavy); Gambhīrapākah (deep seated suppuration), Prājyoṣmā (increased heat), Spṛṣṭaḥ klinnoavadīryate (affected part become very moist and burst out), Paṅkavacchīrṇamāṃsa (the muscles appears like mud and falls off), Spaṣṭasnāyusirāgaṇaḥ (tendons and veins are exposed) and Śavagandhi (emits cadaveric smell).

Sannipātaja Visarpa

- ⊙ सर्वजो लक्षणैः सर्वैः सर्वधात्वतिसर्पणः। (A.H.Ni.13/65)
- ⊙ सर्वात्मकस्त्रिविधवर्णरुजोऽवगाढः पक्वो न सिध्यति च मांससिराप्रशातात् ॥ (Su.Ni.10/6)

The clinical features of Sannipātaja Visarpa are characterized by the combined features of all the three doṣas. Suśruta stated that the Sannipātaja Visarpa has characteristic Varṇa (colour) and Rujā (pain) of all the three doṣas together and it becomes Avagāḍha (deepened). It becomes Asādhya (incurable) when Pāka (suppuration) takes place and Māṃsasiraprasātāt (muscles and vessels are destroyed).

Kṣataja Visarpa

- ⊙ बाह्यहेतोः क्षतात्क्रुद्धः सरक्तं पित्तमीरयन्॥ विसर्पं मारुतः कुर्यात् कुलत्थसदृशैश्चितम्।
  स्फोटैः शोफज्वररुजादाहाढ्यं श्यावलोहितम्॥ (A.H.Ni.13/65-66)
- ⊙ सद्यःक्षतव्रणमुपेत्य नरस्य पित्तं रक्तं च दोषबहुलस्य करोति शोफम् ।
  श्यावं सलोहितमतिज्वरदाहपाकं स्फोटैः कुलत्थसदृशैरसितैश्च कीर्णम् ॥ (Su.Ni.10/7)

Due to the external causes or injury, the aggravated Vāta vitiates the Pitta and Rakta and produces the Kṣataja Visarpa which is associated with Sphoṭaiḥ Kulatthasadṛśa (blisters which resembles the horse gram), Śopha (swelling), Jvara (fever), Rujā (pain), Dāhāḍhyaṃ (burning sensation), Śyāva (blackish) and Salohita (reddish colour). Suśruta described similar symptoms with the difference in the pathogenesis i.e. the involvement of Vāta doṣa is not mentioned.

Upadrava of Visarpa

- ⊙ ज्वरातिसारवमथुत्वङ्गमांसदरणक्लमाः । अरोचकाविपाकौ च विसर्पाणामुपद्रवः ॥ (Y.R.Ci.58/26)

Jvara (fever), Atisāra (diarrhoea), Vamathu (vomiting), Tvaṅmāṃsadaraṇa (tearing of skin and muscles), Klamā (fatigue), Arocaka (anorexia) and Avipāka (indigestion) are the complications of Visarpa.

Sādhyāsādhyata of Visarpa

- ⊙ पृथग्दोषैस्त्रयः साध्या द्वन्द्वजाश्चानुपद्रवाः। असाध्यो क्षतसर्वोत्थौ सर्वे चाक्रान्तमर्मकाः।
  शीर्णस्नायुसिरामांसाः प्रक्लिन्नाः शवगन्धयः। (A.H.Ni.13/67-68)
- ⊙ सिध्यन्ति वातकफपित्तकृता विसर्पाः सर्वात्मकः क्षतकृतश्च न सिद्धिमेति ।
  पैत्तानिलावपि च दर्शितपूर्वलिङ्गौ सर्वे च मर्मसु भवन्ति हि कृच्छ्रसाध्याः ॥ (Su.Ni.10/8)

Visarpa caused by each doṣa separately and those caused by two doṣas without complications are considered as Sādhya (curable); Sannipātaja Visarpa, those which invades vital spots and those producing the Śīrṇasnāyusirāmāṃsā (fall of tendons, veins and muscles), Praklinnā (excessive moist) and emitting Śavagandha (cadaveric smell) are considered as Asādhya (incurable). Suśruta added Kṣataja Visarpa as Asādhya (incurable); Vātaja and Pittaja Visarpa with all the clinical features mentioned before such as blisters in Vātaja and Śīrṇasnāyusirāmāṃsa (fall of tendons, veins and muscles) in Pittaja; and those which situated over the Marma (vital parts) are considered as Kṛcchra Sādhya (difficulty in cure).

In Contemporary science,

Visarpa can be correlated with Erysipelas and Herpes zoster. (Please refer Topic 33)

Short Answer Questions

1. Explain Agni Visarpa
2. Explain Kardama Visarpa
3. Explain Granthi Visarpa
4. Differentiate Doṣaja Visarpa

Long Answer Questions

1. Define Visarpa and explain the Nidāna, Samprāpti, Bheda and Lakṣaṇās of Visarpa

TOPIC 33

Skin Diseases

Introduction

The skin, the largest organ of the human body, acts as a crucial barrier against external elements while regulating body temperature, safeguarding against infections, and enabling sensory perception. However, factors such as genetics, environmental exposures, and lifestyle choices can contribute to the prevalence of skin diseases, impacting millions of individuals globally.

Skin diseases encompass a wide array of conditions, ranging from minor irritations to significant afflictions that profoundly affect physical and emotional well-being. Inflammatory skin diseases include acne, eczema, psoriasis, and rosacea. Infectious skin diseases encompass impetigo, ringworm, warts, herpes simplex, and folliculitis. Autoimmune skin diseases comprise vitiligo, lupus, scleroderma, pemphigus, and alopecia areata. Cancerous skin diseases include melanoma, basal cell carcinoma, squamous cell carcinoma, actinic keratosis, and Kaposi's sarcoma. Genetic skin diseases encompass ichthyosis, epidermolysis bullosa, Ehlers-Danlos syndrome, neurofibromatosis, and porphyria. Allergic skin diseases include contact dermatitis, urticaria, atopic dermatitis, and seborrheic dermatitis. This chapter focuses on diseases such as eczema, urticaria, psoriasis, lichen planus, pemphigus, pemphigoid, leprosy, vitiligo, and cellulitis.

ECZEMA

Introduction

Eczema, also known as dermatitis, is a non-infectious inflammatory skin condition. In its acute stage, the disorder is characterized by intense itching (pruritus), erythema (redness), oedema (swelling), papules, vesicles, oozing, crusting, and, in some cases, blister formation. The subacute stage is distinguished by widespread erythema, oedema, and scaling of the skin. As the condition advances to the chronic stage, it is characterized by persistent itching, hyperkeratosis (thickened skin), and lichenification, which includes thickening, hyperpigmentation, and accentuated skin markings.

The global prevalence of eczema is estimated to be approximately 10-20% of children and 1-3% of adults, with the highest incidence occurring during early childhood. Estimates indicate that one in ten individuals will experience eczema at some point in their lives. In India, the prevalence of eczema is estimated to be between 10.0% and 15.6%.

The causative factors of eczema can be systematically classified into three primary categories: extrinsic, intrinsic, and mixed causes.

Extrinsic causes include allergic reactions to chemical agents, pollens, leaves, fruits, minerals, and physical agents, such as soaps, detergents, fragrances, and contact allergens like metals or latex. Conversely, **intrinsic causes** pertain to allergies arising from specific food articles, gastrointestinal toxins, genetic predisposition, hormonal imbalances, and immunological disorders.

Moreover psychogenic factors including stress, anxiety, and depression can significantly influence the manifestation of the disease. In many instances, eczema arises from a combination of extrinsic and intrinsic factors, influenced further by genetic and environmental elements. Other contributing factors include skin barrier dysfunction, microbial imbalances, and environmental aspects like climate and pollution. Lifestyle factors such as sleep, diet, and exercise also play a crucial role

Pathophysiology

The pathophysiology of eczema involves genetic predisposition, environmental factors, and immune system abnormalities. Individuals diagnosed with eczema frequently exhibit mutations in the filaggrin (FLG) gene, which compromises the integrity of the skin's barrier function. This deficiency renders the skin more susceptible to irritants, allergens, and microbial invasion, ultimately leading to transepidermal water loss and chronic inflammation.

Furthermore, immune dysregulation contributes to an imbalance between T-helper type 2 (Th2) and T-helper type 1 (Th1) immune responses. This imbalance manifests as elevated levels of immunoglobulin E (IgE) antibodies and pro-inflammatory cytokines. Such immunological disturbances precipitate the hallmark symptoms of eczema, which include severe itching, erythema, oedema, and the formation of vesicles and crusts. Persistent scratching exacerbates skin damage, thereby perpetuating a cycle of inflammation and barrier dysfunction.

Classification and Clinical features of Eczema

Eczema is characterized by several distinctive clinical features, which typically include

Pruritus (intense itching), Erythema (redness), Papules (small, elevated bumps), Vesicles or blisters (fluid filled lesions), Crusting or scaling (accumulation of dried exudate), and Skin thickening (hyperkeratosis or increased skin density).

Eczema can be categorized into two primary types based on its underlying causes endogenous and exogenous.

Endogenous eczema is primarily caused by the patient's inherent constitutional factors, with environmental influences also contributing to its development. This condition encompasses a range of specific disorders, including

- **Atopic dermatitis,** also known as infantile eczema, represents the most prevalent form of eczema, predominantly affecting infants around the age of three months. The primary symptoms encompass intense pruritus (itching) and skin lesions that may range from mild erythema (redness) to severe lichenification (thickening of the skin) and erythroderma (widespread redness). This condition typically manifests on the cheeks, scalp, extensor surfaces of the limbs, and trunk. It is characterized by dry, scaly skin and an increased susceptibility to skin infections. Atopic dermatitis frequently presents a family history of atopy, including asthma and allergies. The condition progresses through three distinct phases:
  - **Infantile phase** (0-2 years) affects the face, neck, and extensor surfaces
  - **Childhood phase** (2-12 years) affects flexural surfaces such as the elbows and knees
  - **Adult phase** (after 12 years) involves the hands, feet, and face.
- **Seborrhoeic dermatitis** is characterized by inflammation and flaking skin, typically affecting the scalp, as well as other oily areas such as the face, ears, neck, and chest. Symptoms include constant itching, reddish-yellow or pink rash, flaky, greasy, or crusty skin, yellowish or brownish patches. In severe cases, this condition may also lead to hair loss.
- **Nummular eczema,** also known as discoid eczema or nummular dermatitis, is characterized by the presence of scattered circular patches on the skin. These coin-shaped patches, typically measuring 1-10 cm in diameter, commonly appear on the legs, arms, torso, and buttocks. They may present as oozing or extremely dry and sensitive, often accompanied by itching, burning, or pain.
- **Pompholyx,** also known as Dyshidrotic eczema, is characterized by the appearance of small, fluid-filled blisters on the palms of hands, soles of feet, and edges of fingers and toes. This condition, frequently referred to as hand eczema or foot eczema, primarily affects adults aged 20 to 40 years and is more prevalent among males. The blisters, generally measuring 1-5 mm, are typically itchy and may be accompanied by redness, swelling, crusting, and scaling.

- **Asteatotic eczema,** also known as eczema craquele or dry eczema, is characterized by excessive dryness of the skin, often occurring during the winter months or in dry environments. This type of eczema typically affects the lower legs, arms, and torso, leading to the formation of fine cracks and fissures in the skin's surface. Symptoms may include intense itching, redness, inflammation, and scaling.
- **Stasis dermatitis,** also known as gravitational dermatitis, venous eczema, or venous stasis dermatitis, arises due to poor circulation in the legs. This condition results from increased venous pressure and fluid accumulation in weakened veins, leading to inflammation and potential skin damage. Symptoms typically manifest in the affected leg and may include itchy skin, varying degrees of redness (red in lighter skin tones and brown, purple, gray, or ash in darker skin tones), dryness, swelling, varicose veins, and, in severe cases, ulcerations.
- **Juvenile plantar dermatitis** is a skin condition affecting children and adolescents, typically between 3-14 years. It results in inflammation and irritation on the soles of the feet, with symptoms including redness and scaling, cracking and fissuring, itching or burning sensations, dryness and tightness, and painful or tender skin. This condition is often associated with overuse or repetitive friction, inadequate footwear, moisture buildup, genetic predisposition, and allergies or sensitivities..
- **Lichen simplex chronicus,** also known as neurodermatitis, is characterized by thickened, scaly, and intensely itchy patches. These patches typically appear on the arms, legs, scalp, or genital area due to repetitive scratching or rubbing, resulting in skin thickening and inflammation. Symptoms include intense itching and scratching, thickened skin patches (lichenification), a scaly or crusty texture, redness and inflammation, and hyperpigmentation.

Exogenous eczema constitutes a category of dermatitis arising from the skin's exposure to external irritants or toxic agents. It encompasses conditions such as:

- **Contact dermatitis** is characterized by inflammation and irritation following exposure to an external substance. It can be classified into two main types: **Irritant Contact Dermatitis** (ICD), caused by harsh substances like chemicals, detergents, and solvents, and **Allergic Contact Dermatitis** (ACD), triggered by allergens such as latex, metals, and fragrances. Symptoms similar to eczema, including redness, itching, burning, dryness, cracking, swelling, blistering, and rash.
- **Photodermatitis,** also known as sun poisoning or solar dermatitis, is induced by an adverse reaction to sunlight. It results in inflammation, blisters or rashes, itching, burning or stinging sensations, and dryness and cracking of the skin, typically in sun-exposed areas. This condition may arise from excessive sun exposure, sensitivity to particular medications, contact with photosensitizing plants, or underlying skin conditions such as lupus or porphyria.
- **Infectious eczemoid dermatitis** is a rare dermatological condition characterized by

an eczema-like reaction triggered by an infectious agent, commonly observed among individuals with pre-existing skin conditions or those with compromised immune systems. The etiological factors include bacterial infections, notably Staphylococcus aureus and Streptococcus pyogenes; viral infections, such as herpes simplex and varicella-zoster; and fungal infections, including Candida albicans. Symptoms manifest as redness, swelling, and inflammation, accompanied by vesicles, crusts, or pustules, itching, burning, or stinging, and skin thickening and lichenification.

Diagnosis

Diagnosis is typically based on clinical evaluation, including medical history, physical examination, and family history. Diagnostic criteria include pruritus (itching), erythema (redness), papules or vesicles, crusting or scaling, and skin thickening (lichenification). Diagnostic tests such as skin scraping or biopsy, patch testing for contact dermatitis, allergy testing for atopic dermatitis, skin prick tests, and blood tests to rule out underlying conditions.

URTICARIA

Introduction

Urticaria, also known as hives or wheals, is a skin condition characterised by the sudden onset of itchy, raised, and oedematous plaques or welts. These lesions can vary in size, shape, and colour and typically resolve within 24 hours. Causative factors include allergic reactions to food, insect stings, medications, and latex, as well as hypersensitivity reactions, comprising immediate (Type I) and delayed (Type IV) hypersensitivity. Other triggers encompass infections (bacterial, viral, parasitic), autoimmune disorders (lupus, rheumatoid arthritis, thyroid disease), physical stimuli (cold, heat, pressure, vibration), stress, genetic predisposition, hormonal changes (thyroid dysfunction, pregnancy, menstruation), and medications (antibiotics, NSAIDs, anesthetics).

As of 2017, the global prevalence of urticaria was estimated to be around 86 million individuals, representing approximately 1.1% of the worldwide population. In India, the prevalence of urticaria is reported to be around 0.1% of the population, with an incidence of about 15%. The condition is more prevalent within the 20-40 years old, although its incidence is higher among older age groups.

Pathophysiology

The pathophysiology of urticaria involves an intricate interplay of immune cells and inflammatory responses. with hypersensitivity reactions triggering mast cell activation and chemical mediator release, including histamine, bradykinin, and kallikrein, which are stored in granules within the cells. IgE antibodies facilitate the release of these mediators, playing a critical role in allergic urticaria. Histamine, bradykinin, and kallikrein induce vasodilation and increased permeability of blood vessels in the skin, leading to fluid leakage and the formation of raised, red, and pruritic wheals.

Classification and Clinical features:

The clinical features of urticaria include wheals with a raised, flat-topped appearance surrounded by a red flare, angioedema affecting the face, lips, tongue, or throat, and dermatographism, where skin reacts to minor trauma with wheal formation. Symptoms encompass intense itching or burning, skin tenderness, swelling, and redness, with flares triggered by stress, temperature, or physical activity. Urticaria is categorized into two primary types: acute and chronic.

- **Acute urticaria** manifests abruptly and endures for less than six weeks, often due to allergic responses (e.g., food, insect stings, and medications), infections (bacterial, viral, parasitic), stress, and environmental stimuli (e.g., cold, heat, sunlight). It is characterized by variable-sized wheals with a red flare, often accompanied by angioedema, or swelling of deeper skin layers.
- **Chronic urticaria,** persisting for more than six weeks, is frequently associated with autoimmune disorders such as lupus, rheumatoid arthritis, SLE, Sjogren syndrome, autoimmune thyroid disease, endocrine irregularities including thyroid dysfunction and elevated progesterone levels, and genetic predisposition. It manifests with recurring wheals, persistent itching, and discomfort.

Urticaria types include spontaneous (non identifiable cause), inducible (triggered by specific stimuli), autoimmune (associated with autoimmune disorders), and allergic (triggered by allergens). Complications may arise, such as anaphylaxis (life-threatening allergic reaction), angioedema (airway obstruction or gastrointestinal involvement), and skin infections (secondary bacterial infections).

Diagnosis:

Diagnosis is based on comprehensive clinical evaluation, laboratory tests and Immunological tests. The clinical evaluation involves detailed history taking and physical examinations to identify the underlying cause, severity, and duration of symptoms. This includes assessing the onset, frequency, and triggers of episodes, along with relevant medical and family histories. The physical examination focuses on skin assessment for lesion characteristics, vital signs, dermatographism, and angioedema.

Laboratory investigations may reveal abnormalities, though normal results do not rule out a diagnosis. Haematological tests can indicate elevated eosinophil and leukocyte counts, anaemia, and thrombocytosis. Biochemical tests might show elevated erythrocyte sedimentation rate (ESR), increased C-reactive protein (CRP), liver enzyme elevation, and renal impairment.

Immunological tests may detect positive antinuclear antibodies (ANA), elevated immunoglobulin E (IgE) levels, and autoantibodies such as anti-thyroid antibodies. Allergy testing methods, including skin prick testing (SPT), radioallergosorbent test (RAST), and enzyme-linked immunosorbent assay (ELISA), assist in identifying specific allergens. Infectious disease tests may confirm bacterial, viral, or parasitic infections, while autoimmune tests such as thyroid function tests and rheumatoid factor (RF) assessments aid in diagnosing underlying conditions.

PSORIASIS

Introduction

Psoriasis is a chronic autoimmune inflammatory skin disorder characterized by brownish-red papules and plaques that are sharply demarcated and are covered with fine, silvery white scales. These lesions predominantly manifest on extensor surfaces, the scalp, and the lumbosacral region. Globally, approximately 125 million individuals are affected by psoriasis, representing about 2-4% of the total population. In India, the prevalence of psoriasis varies between 0.4% and 2.5%. This condition is observed to be more prevalent in males, particularly during their third or fourth decade of life.

Pathophysiology:

The pathophysiology of psoriasis is characterized by a multifactorial interplay of genetic, immunological, environmental, and cellular elements. Genetic predisposition to psoriasis is notably linked to mutations, primarily in the HLA-Cw6 allele, which initiates an autoimmune response. In this response, dendritic cells in the skin present antigens to T-cells, particularly involving Th17 cells. These Th17 cells secrete pro-inflammatory cytokines, including IL-17, IL-22, and TNF-α, which drive the inflammatory process. The release of these cytokines induces excessive proliferation of keratinocytes, resulting in the development of thick, scaly plaques. The interaction among keratinocytes, dendritic cells, T-cells, and endothelial cells sustains the cycle of inflammation and cellular proliferation. The accelerated turnover of skin cells inhibits their maturation, thereby producing the characteristic scales associated with psoriasis.

Environmental factors such as stress, infections, trauma, winter season, alcohol consumption, smoking, obesity, and specific medications including chloroquine, lithium, steroids, and beta-blockers etc., can exacerbate the condition. These triggers activate immune cells, leading to an elevated production of cytokines and subsequent inflammation.

Classification and Clinical features:

Psoriasis is characterized by well-defined, erythematous plaques that are scaly and thickened, often accompanied by itchiness or pain. These plaques, often covered with silvery-white or yellowish scales, commonly manifest on the elbows, knees, scalp, and lower back. Psoriasis can be categorized into several distinct types based on its clinical presentation.

- **Plaque psoriasis,** also known as psoriasis vulgaris or chronic plaque psoriasis, represents the most prevalent form of the condition, accounting for approximately 90% of cases. It is distinguished by discrete, erythematous plaques with silvery scales, primarily located on the extensor surfaces of the extremities, such as the elbows, knees, scalp, and back.
- **Guttate psoriasis** also known as eruptive psoriasis is frequently observed in children and young adults, especially following streptococcal pharyngitis. This condition is characterized by the sudden onset of multiple erythematous plaques measuring between 0.5 and 1.5 cm in diameter, exhibiting a raindrop-shaped appearance. The scaly lesions are predominantly located on the trunk and back.
- **Pustular psoriasis** is characterised by the formation of small non-infectious pustules surrounded by erythema. It is of two types localized and generalized.
    - **Localized pustular psoriasis** is restricted to specific areas, such as the hands and feet, and typically resolves without leading to systemic complications.
    - **Generalized pustular psoriasis** represents a more severe manifestation, presenting with sterile pustules on widespread erythematous plaques across the entire body. This form is often associated with additional conditions, including geographic tongue and hypocalcaemia.
- **Erythrodermic psoriasis** also known as exfoliative psoriasis represents a severe and potentially life-threatening form of psoriasis. This condition is characterized by widespread inflammation and exfoliation, affecting more than 90% of the body surface area. The onset is often sudden and manifests with extensive erythema, exfoliation, scaling, intense pruritus, burning sensations, swelling, oedema, fever, and general malaise. Erythrodermic psoriasis frequently arises from the exacerbation of unstable plaque psoriasis; with the abrupt discontinuation of systemic corticosteroids is a common triggering factor. Complications of erythroderma psoriasis may include impairment in barrier functions of the skin, disturbance in basal metabolic rate, and increased cutaneous circulation, which can adversely affect cardiac function and potentially lead to cardiac failure.
- **Inverse psoriasis,** also known as flexural psoriasis or intertriginous psoriasis is characterized by smooth, erythematous, and distinctly defined patches that primarily affect intertriginous areas such as inguinal, gluteal, axillary, inframammary, and retroauricular folds as well as the glans of the uncircumcised penis. The affected skin appears moist, macerated, and may contain fissures, often accompanied by a foul odor and itching or pain. Notably, inverse psoriasis lacks scales, which differentiates it from other psoriasis variants..

- **Palmoplantar psoriasis** is characterised by red, scaly, and cracked patches, as well as thickening of the skin (hyperkeratosis), which can include pustules or vesicles on the palms of the hands and soles of the feet. This condition is often accompanied by itching and burning sensations. There are three main types:
    - **Palmoplantar plaque psoriasis:** This type is marked by red, scaly, and thickened patches on the palms and soles, featuring well-defined borders. It often leads to dry, cracked skin, along with itching or burning sensations.
    - **Palmoplantar pustular psoriasis:** This form presents with small pustules on the palms and soles. It may also show yellowish or brownish crust formation, accompanied by redness, swelling, and potentially fever or malaise.
    - **Palmoplantar eccrine psoriasis:** This type involves the eccrine glands (sweat glands) and leads to vesicles or pustules on the palms and soles, accompanied by burning or stinging sensations, redness, swelling, and excessive sweating (hyperhidrosis).
- **Scalp psoriasis** is characterized by red, scaly, and inflamed patches on the scalp. It is often associated with dandruff-like flaking, crusty or powdery scales, and uncomfortable sensations such as itching, burning, or stinging. In some cases, it can result in temporary or permanent hair loss.
- **Nail psoriasis** is characterised by distinct features, including pitting, thickening of the nail plate, subungual hyperkeratosis, onycholysis, and oil spots. This condition can affect both fingernails and toenails, often occurring alongside skin psoriasis, though it can also manifest alone.

Complications

Psoriasis can result in a variety of complications that encompass both physical and psychological aspects. Physically it can cause psoriatic arthritis, eye-related conditions, including conjunctivitis, uveitis, blepharitis, and dry eye syndrome, and increased risk of cardiovascular diseases, cerebrovascular accidents (strokes), and metabolic syndrome, which includes conditions such as obesity, diabetes, and hypertension. Additionally, the compromised skin barrier increases the risk of infections. In severe manifestations, such as erythrodermic psoriasis, respiratory complications may occur. Psychologically, psoriasis often leads to mental health challenges like anxiety, depression, and social isolation.

Diagnosis

Diagnosis is based on clinical evaluation, dermatological tests, and skin biopsy. Clinically, a family history of the condition, alongside the manifestation of characteristic symmetrical skin lesions such as plaques, scales, and erythema located on the scalp, elbows, knees, and lower back, is suggestive of psoriasis. Important diagnostic indicators include the Auspitz sign, which is characterized by multiple bleeding points upon skin scraping, and the Koebner phenomenon, where lesions appear at sites of trauma.

Dermatological tests like KOH (Potassium hydroxide test) and Wood's lamp examination help exclude the possibility of fungal or bacterial infections. A skin biopsy provides definitive confirmation of psoriasis and helps differentiate it from other dermatological conditions, including eczema, seborrheic dermatitis, and fungal infections.

LICHEN PLANUS

Introduction

Lichen planus is a chronic inflammatory condition affecting the skin, mucous membranes, hair follicles, and nails. It is characterized by small, shiny, violaceous, flat-topped, and pruritic polygonal papules, typically affecting the wrists, shins, trunk, medial thighs, and glans penis. Globally, the prevalence ranges from 0.5% to 5%, while in India, it varies between 0.44% and 4.79%. Lichen planus is more common in females than males, primarily affecting individuals between 40-60 years of age.

Pathophysiology

The pathophysiology of Lichen planus remains incompletely understood, yet it involves a complex interaction among immune, genetic, and environmental factors. This autoimmune disorder is characterized by the targeting of basal keratinocytes by T cells, specifically CD4+ and CD8+ cells. The resultant immune response triggers the release of cytokines, including interferon-gamma and tumor necrosis factor (TNF)-alpha, which lead to the apoptosis of basal keratinocytes and the formation of distinctive lesions.

Additionally, chronic inflammation is perpetuated by the activity of dendritic cells, macrophages, and mast cells. Contributing factors may include genetic predisposition, psychological stress, infections such as hepatitis C, certain medications, and exposure to allergens. The immune response and accompanying inflammation culminate in lesions that exhibit hyperkeratosis, acanthosis, and a band-like infiltrate of lymphocytes. Clinically, Lichen Planus manifests as pruritic papules, skin lesions, and mucosal involvement.

Classification and Clinical features

Lichen planus is clinically characterised by small, shiny, violaceous, flat-topped, and pruritic polygonal papules. They are usually symmetrically distributed, most commonly on the flexor surfaces of the wrists, legs, trunk, glans penis, and oral and vaginal mucosae but can be widespread. The face is rarely involved.

Lichen planus is clinically classified into several types.

- **Cutaneous Lichen planus** affects the skin, presenting with pruritic, polygonal papules on flexor surfaces, trunk, and genitalia.

- **Oral Lichen planus** involves mucous membranes, exhibiting white, lacy or reticular patches, linear white or gray wickham striae, erosive lesions, and red, atrophic areas on the buccal mucosa, tongue, lips, gingiva, oesophagus and larynx.
- **Genital Lichen planus** affects genitalia, similar to cutaneous Lichen planus.
- **Lichen Planopilaris (LPP)** causes scarring alopecia on the scalp.

Rare variants include

- **Actinic Lichen planus,** with annular lesions in sun-exposed areas;
- **Annular Lichen planus,** featuring ring-shaped lesions;
- **Atrophic Lichen planus,** characterized by thin skin;
- **Bullous Lichen planus,** with blistering lesions;
- **Hypertrophic Lichen planus,** displaying thickened skin; and
- **Linear Lichen planus,** presenting with linear or zosteriform lesions.

Diagnosis

Diagnosis is based in clinical evaluation and biopsy. Physical examination reveals the characteristic features, often summarized by the "six Ps": pruritic (itchy), polygonal (many sharp angles), planar (flat-topped), purple, papules (bumps), and plaques. Skin or mucous membrane biopsy confirm the diagnosis and helps exclude other conditions such as lichenoid dermatitis, psoriasis, discoid lupus erythematosus, pemphigus, and aphthous stomatitis.

PEMIPHIGUS

Introduction

Pemphigus is a group of chronic autoimmune bullous diseases characterized by the development of blisters, approximately 1 cm in diameter, along with erosions affecting both the skin and mucous membranes. This condition predominantly manifests as painful blisters and erosions within the oral cavity. Skin involvement can present with variability, ranging from localized to extensive lesions. The global incidence of pemphigus among dermatology outpatients is estimated to be between 0.09% and 1.8%. In India, the incidence is approximately 4.4 cases per million individuals annually.

Pathophysiology

The pathophysiology of Pemphigus involves the formation of autoantibody against desmogleins (Dsg1 and Dsg3), which are essential adhesion molecules in the skin. These autoantibodies trigger an immune response, activating the complement system and T-cell mediated immunity, leading to inflammation and tissue damage. Consequently, keratinocyte adhesion is disrupted, causing intraepidermal blister formation.

Furthermore, various signaling pathways regulate the release of cytokines and apoptosis, contributing to the development of intraepidermal blisters and acantholysis. Both genetic predisposition and environmental triggers such as infections and UV radiation, play significant roles in the disease development. The concepts of desmoglein compensation and epitope spreading provide additional insight into the complex interactions that characterize pemphigus.

Classification and Clinical Features:

Pemphigus is characterized by the presence of flaccid blisters and erosions affecting the skin and mucous membranes. Common clinical manifestations include painful oral lesions, skin lesions located on the trunk, extremities, and face, as well as involvement of mucous membranes in areas such as the oral cavity, genitals, and the conjunctival and nasal mucosa. The lesions associated with this condition are typically flaccid, easily ruptured, and crusted.

The clinical classification of pemphigus encompasses several distinct types:

- **Pemphigus Vulgaris:** This is the most prevalent form, affecting both the skin and mucous membranes, and is characterized by flaccid blisters, erosions, and ulcers.
- **Pemphigus Foliaceus:** This variant is restricted to the skin and is identified by superficial, flaccid blisters accompanied by crusted erosions.
- **Pemphigus Erythematosus:** This form combines characteristics of both Pemphigus Vulgaris and Pemphigus Foliaceus, impacting the skin and mucous membranes.
- **Paraneoplastic Pemphigus:** This rare type is linked to underlying neoplasms and presents severe, widespread lesions affecting both the skin and mucous membranes, including the eyes, mouth, nasopharynx, and oesophagus.
- **Pemphigus Vegetans:** This is characterized by vegetating lesions and predominantly affects intertriginous areas of the body.

Diagnosis:

Diagnosis is based on clinical evaluation, biopsy, histopathology, immunofluorescence studies, and autoantibody testing. Clinically, the condition presents with flaccid blisters, erosions, and ulcers on the skin and mucous membranes. Biopsies of skin and mucosal tissues typically show intraepidermal blisters and acantholysis.

Histopathology reveals these intraepidermal blisters, acantholysis, and an intact basal cell layer. Immunofluorescence studies, including both direct and indirect methods, are used to detect autoantibodies. Additionally, autoantibody testing through techniques such as ELISA and immunoblotting confirms the presence of autoantibodies against desmogleins.

PEMPHIGOID

Introduction

Pemphigoid is a group of rare autoimmune bullous diseases characterized by the formation of large, tense blisters, generally exceeding 1 cm in diameter, accompanied by erosions affecting both the skin and mucous membranes. In contrast to Pemphigus, Pemphigoid blisters exhibit a reduced propensity to rupture due to their tense structure, resulting in relatively intact blisters without the occurrence of acantholysis.

The estimated global incidence of Pemphigoid ranges from 2.4 to 23 cases per million individuals in the general population. In India, while specific prevalence data are limited, studies indicate that autoimmune bullous diseases, including Pemphigoid, are comparatively infrequent. This condition demonstrates a higher prevalence in females than in males and is particularly noted among individuals aged 70 years and older.

Pathophysiology

The pathophysiology of Pemphigoid involves an autoimmune response triggered by genetic predisposition and environmental factors leading to the production of autoantibodies against bullous pemphigoid antigen 230 (BP230) and bullous pemphigoid antigen 180 (BP180) in the basement membrane zone (BMZ). This initiates a cascade of immune responses, activating complement and inflammatory pathways, resulting in neutrophil and eosinophil infiltration, tissue damage, and blister formation. Cytokines (IL-1β, TNF-α) amplify inflammation, while T-cell activation and cytokine release contribute to antibody-dependent cellular cytotoxicity (ADCC) and complement-mediated damage. Ultimately, BMZ disruption separates the epidermis from the dermis, allowing fluid accumulation and blister formation.

Classification and Clinical features

Pemphigoid is characterized by the presence of large, tense blisters, typically exceeding 1 cm in diameter. These lesions often appear on the trunk, extremities, oral mucosa, genital mucosa, and conjunctiva. They are often accompanied by surrounding skin exhibiting erythematous, urticarial, or oedematous characteristics. The blisters and erosions associated with this condition may heal with resultant scarring and post-inflammatory hyperpigmentation. Mucosal involvement can present as erosions, ulcers, and bleeding, particularly affecting the oral cavity, while involvement of the genital mucosa and conjunctiva is also prevalent. Additional symptoms may include pruritus, pain, heightened skin sensitivity, as well as systemic manifestations such as weight loss and fever.

The clinical classification of Pemphigoid consists of several distinct types

- **Bullous Pemphigoid:** This is recognized as the most prevalent form, characterized by large, tense blisters on the skin, frequently accompanied by pruritus. It primarily affects the elderly population.
- **Mucous Membrane Pemphigoid:** This variant primarily affects mucous membranes, resulting in erosions, ulcers, and scarring in the oral cavity, eyes, nose, throat, and genital regions, often in association with systemic diseases..
- **Pemphigoid Gestationis:** This rare form occurs in association with pregnancy, typically during the second or third trimester. It is characterized by intense pruritus and blistering, often commencing around the umbilicus.
- **Linear IgA Bullous Dermatosis:** This condition is defined by linear IgA deposits at the basement membrane zone, leading to blistering lesions that are frequently triggered by medications or infections.

Diagnosis

Diagnosis is based on clinical evaluation, biopsy, immunofluorescence studies, and autoantibody testing. Clinically, Pemphigoid is characterized by the presence of large, tense blisters, accompanied by itching and mucosal involvement. A skin biopsy is instrumental in confirming the existence of subepidermal blisters and assessing the involvement of the basement membrane zone.

Direct immunofluorescence tests are utilized to detect IgG and C3 autoantibodies within the skin, whereas indirect immunofluorescence tests identify circulating autoantibodies that target components of the basement membrane zone. Furthermore, serum autoantibody testing serves to confirm the presence of autoantibodies against BP230 and/or BP180.

MYCOTIC SKIN DISEASE

Introduction

Mycotic skin diseases, also known as fungal skin infections, constitute a range of conditions caused by pathogenic fungi that infect the skin, hair, and nails. These infections occur when fungi, typically found in the environment, enter the body through cuts, abrasions, or other openings in the skin. Globally, fungal skin diseases affect more than a billion people and are responsible for over 1.5 million deaths annually. In India, the prevalence of fungal infections is notably high, with an estimated 57 million people affected by fungal skin diseases.

Several factors can increase the risk of developing these infections, including Poor hygiene, warm and humid environments, skin injuries, weakened immunity, contaminated water or soil, and close contact with infected individuals.

Pathophysiology

The pathophysiology of Mycotic skin diseases involves the invasion and colonization of the skin by pathogenic fungi. Fungi enter the skin through minor cuts or abrasions, adhering to skin cells via specialized adhesins and invasins. Upon attachment, they secrete enzymes that break down the skin's protective barrier, facilitating deeper penetration and dissemination.

As the fungal infection advances, the immune system initiates a response characterized by inflammation and the activation of various immune cells, including neutrophils, macrophages, and T-cells. However, in individuals with compromised immune function, the fungi may evade this immune response, resulting in unchecked proliferation and potentially severe infections. The fungal invasion can also induce morphological alterations in the skin, such as epidermal hyperplasia, dermal inflammation, and vascular dilation. Furthermore, in instances of deep mycoses, the infection may disseminate to other tissues and organs, leading to systemic complications.

Classification and Clinical features:

- Mycotic skin diseases present with a variety of clinical features, including redness and inflammation, itching and burning sensations, skin lesions, rashes or ulcers, nail discolouration or thickening, and hair loss.
- Mycotic skin diseases are categorized into three main types: superficial, subcutaneous, and deep mycoses.
- **Superficial Mycoses:** Fungal infection affects skin, nails, and mucous membranes. Examples include:
  - **Dermatophytosis (Tinea / Ringworm)** is a fungal infection of the skin that may affect skin, hair, and nails. It is characterized by a red, itchy, scaly, circular rash and hair loss at the affected site. The infection can occur in various regions of the body, including.
    - **Tinea pedis (athlete's foot):** Affects the feet, often causing itching, burning and cracked skin between the toes.
    - **Tinea cruris (jock itch):** Affects the groin and inner thighs, leading to red, itchy, and often ring-shaped rashes.
    - **Tinea capitis:** Affects the scalp, resulting in scaly patches and hair loss.
    - **Tinea manuum:** Affects the hands, causing red, itchy, and sometimes painful rashes.
    - **Tinea barbae:** Affects facial hair and the surrounding skin, leading to red, itchy patches and sometimes pustules.
    - **Tinea corporis:** Refers to infections in other areas of the body, presenting as red, itchy, circular rashes with clear centres

  - **Onychomycosis** is a fungal infection of fingernails or toenails, causing discolouration and structural damage.
  - **Candidiasis** is a fungal infection caused by Candida albicans, affecting the skin and mucous membranes, manifesting as oral thrush, diaper rash, vaginal yeast infections (vulvovaginitis), oesophageal candidiasis, and candidal intertrigo.
  - **Tinea Versicolor (pityriasis versicolor):** Skin condition caused by Malassezia fungus, resulting in alterations in skin pigmentation, discolouration (hypopigmentation or hyperpigmentation) and scaly patches.
- **Subcutaneous Mycoses:** Fungal infection affects the subcutaneous tissues through cuts or wounds, causing rashes, ulcers, and other symptoms on the skin. Examples include
  - **Sporotrichosis,** also known as Rose Gardener's Disease, is caused by the Sporothrix fungus. It typically affects the skin but can also infect the lungs and other parts of the body.
  - **Chromoblastomycosis** is caused by various fungi, including Fonsecaea and Phialophora, and leads to chronic skin lesions and warty growths.
  - **Eumycetoma** is caused by fungi such as Madurella and Nocardia. It primarily affects the feet and is characterized by swelling, pain, discharge, and the formation of sinuses and granules.
- **Deep Mycoses:** Fungal infection disseminates through the bloodstream, affecting multiple organs. Examples include
  - **Cutaneous Histoplasmosis** is a fungal infection caused by inhaling Histoplasma capsulatum spores, and it can manifest on the skin in rare cases. It typically presents as skin lesions or ulcers, papules or nodules, abscesses or cellulitis, and erythema nodosum (red, painful nodules). In severe cases, disseminated histoplasmosis can spread to the skin, causing widespread lesions, oral or genital ulcers, and erythema multiforme (skin and mucous membrane lesions).
  - **Cutaneous Aspergillosis** is a fungal infection of the skin and subcutaneous tissue, typically occurring through direct inoculation of Aspergillus spores. It typically presents as skin lesions or ulcers, redness, swelling and warmth around affected areas, pain or tenderness, pus or discharge and fever.

Diagnosis

Diagnosis is based on clinical examination, laboratory tests, and imaging studies. Clinical examination includes assessing the patient's medical history, performing a physical examination, and using specialized dermatological tools like dermatoscopes to examine the skin and hair closely.

Laboratory tests involve skin scrapings treated with potassium hydroxide (KOH) solution to dissolve cells and enable microscopic examination for fungal elements. Additional tests include biopsies for histopathological examination and cultures to grow fungal organisms, identify species, and determine susceptibility to antifungal agents. Molecular tests, such as PCR and DNA sequencing, are used to detect fungal DNA in skin samples, providing precise identification of specific fungal species. Imaging studies, including X-rays, computed tomography (CT) scans, magnetic resonance imaging (MRI), and ultrasound, evaluate bone involvement, soft tissue involvement, and the depth and extent of lesions.

LEPROSY

Introduction

Leprosy, also known as Hansen disease is a chronic granulomatous infection caused by acid-fast bacilli from the Mycobacterium leprae complex, which includes Mycobacterium leprae and Mycobacterium lepromatosis. This disease primarily affects the skin and peripheral nerves, resulting in distinct lesions, numbness, and sensory impairment. The World Health Organization (WHO) identifies it as a significant public health issue, especially in tropical and subtropical regions. In the year 2021, there were 140,311 new cases of leprosy reported globally, resulting in a prevalence rate of 0.24 cases per 10,000 individuals. India alone accounted for 86,663 of these new cases, representing over 60% of the total global incidence.

The incubation period of leprosy, defined as the interval between exposure to Mycobacterium leprae and the onset of symptoms, typically ranges from 3 to 5 years. However, this period may vary from 1 to 20 years, and in some instances, it may extend beyond 30 years.

Several risk factors increase susceptibility to leprosy, including age (peak incidence occurs between 15 and 45 years), male gender, poverty, close contact with infected individuals, and specific occupations. Environmental factors such as residing in endemic areas, inadequate sanitation, and malnutrition also contribute to transmission. Individuals with weakened immune systems, genetic predispositions, prior exposure, and those living in crowded conditions face increased risk. Vulnerable populations include children under 15, pregnant women, and the malnourished.

Pathophysiology

The pathophysiology of leprosy is characterized by the invasion of Mycobacterium leprae, primarily occurring through respiratory droplets resulting from prolonged close contact with an infected individual. Transmission may also occur via skin-to-skin contact and potentially through contaminated water sources. Upon entry into the host, these bacteria primarily target the skin and peripheral nerves, eliciting a complex immune response.

Mycobacterium leprae invades Schwann cells, macrophages, and dendritic cells, leading to nerve demyelination, axonal damage, and the formation of granulomas. This results in numbness, sensory loss, muscle weakness, and paralysis in severe cases, along with skin lesions, disfigurement, and potential disability.

The immune response determines the clinical presentation of the disease. In cases of tuberculoid leprosy, which is characterized by robust cell-mediated immunity, symptoms are typically localized with limited nerve damage. Conversely, lepromatous leprosy is marked by significant bacterial dissemination and substantial tissue damage, arising from diminished cell-mediated immunity and increased humoral immunity. Furthermore, the lipid envelope of the bacteria facilitates their entry into host cells, while the cytokine profile of the immune response significantly influences disease progression.

Classification and Clinical features

Leprosy should be suspected in individuals presenting with specific symptoms and signs, as indicated by the World Health Organization (WHO). Key indicators include:

- Pale or reddish patches on the skin
- A loss or reduction of sensation in the affected skin areas
- Numbness or tingling sensations in the hands and feet
- Painful or tender nerves
- Swelling or nodules located on the face or earlobes
- Painless wounds or burns on the hands or feet
- Additional signs that may suggest the presence of leprosy encompass:
- Enlarged peripheral nerves
- Muscle weakness or atrophy
- Inflammation of the eyes or loss of vision
- Nasal congestion or respiratory difficulties
- Involvement of the testicles or larynx

Leprosy is classified into two main forms namely Paucibacillary and Multibacillary Leprosy, depending on the type and number of skin areas affected.

- **Paucibacillary Leprosy (PB leprosy)** is characterized by a lower bacterial load, presenting with a limited number of skin lesions (less than five), localized nerve damage, and generally milder symptoms. Clinically, it is characterized by one or a few skin lesions, numbness or tingling in the affected regions, and muscle weakness.
- **Multibacillary Leprosy (MB leprosy)** is characterized by a higher bacterial overload,

multiple skin lesions (more than five), extensive nerve damage, and more severe symptoms. This form of leprosy typically exhibits a multiple skin lesions, widespread numbness or tingling, muscle weakness, enlarged lymph nodes, and disseminated bacterial infection.

The Ridley-Jopling Classification system categorizes leprosy into five distinct forms based on clinical, bacteriological, and immunological criteria.

- **Tuberculoid Leprosy (TT):** This form is characterized by the presence of one to five well-defined, hypopigmented lesions exhibiting clear boundaries and raised margins. The lesions are often scaly and may present as anesthetic patches on the skin. Nerve involvement is common and can lead to significant nerve damage and muscle weakness. Due to the presence of a limited bacterial load and robust cell-mediated immunity, this form generally has a good prognosis.
- **Borderline Tuberculoid Leprosy (BT):** This form is similar to Tuberculoid Leprosy but features a greater number of lesions, typically ranging from five to ten, which are less well-defined. Nerve involvement remains prevalent, accompanied by moderate bacterial activity and moderate cell-mediated immunity, indicating an unstable immune response.
- **Mid-Borderline Leprosy (BB):** This highly unstable form of leprosy is characterized by fluctuating immunity and exhibits traits of both Tuberculoid and Lepromatous Leprosy. Patients commonly present with numerous lesions (exceeding ten) that are irregular in shape, along with significant nerve involvement, resulting in an uncertain prognosis.
- **Borderline Lepromatous Leprosy (BL):** This variant resembles Lepromatous Leprosy, manifesting as erythematous macules, nodules, or papules that lack a distinct pattern. The lesions are often poorly defined, contain a substantial bacterial load, and are associated with weakened immunity.
- **Lepromatous Leprosy (LL):** This form is marked by the presence of more than twenty symmetric skin lesions, which may include nodules, plaques, and papules. Extensive nerve involvement leads to sensory loss and muscle weakness. Additional symptoms can include thickened skin, loss of body hair, nodular enlargement of the earlobes, nasal congestion, ocular complications, and involvement of the testicles and larynx. Lepromatous leprosy is characterized by a high bacterial load and diminished cell-mediated immunity, resulting in a poor prognosis.

Diagnosis

Diagnosis is based on clinical, bacteriological, and immunological evaluations. Clinically, leprosy is identified by the presence of characteristic skin lesions, nerve damage, and other symptoms. A detailed medical history and comprehensive physical examination are essential, including an assessment of skin lesions, nerve function, and sensory impairment.

Bacteriological diagnosis involves detecting Mycobacterium leprae in skin smears or biopsy samples. Skin smears are taken from suspected lesions and stained with Ziehl-Neelsen stain to visualize the bacteria. Biopsy specimens are analyzed for histopathological alterations and the presence of bacterial entities. Immunological tests, such as the lepromin test, assess cell-mediated immunity against M. leprae. Furthermore, molecular diagnostic methods, including polymerase chain reaction (PCR), are employed to identify bacterial DNA in clinical samples.

VITILIGO

Introduction

Vitiligo is a chronic autoimmune skin disorder that is defined by the destruction of pigment-producing cells, referred to as melanocytes. This leads to the development of white patches or spots on the skin. Approximately 1% of the global population is affected by this condition; however, in India, the prevalence can range from 0.1% to more than 8.8%, irrespective of age, sex, or ethnicity.

The exact causes of Vitiligo are remains unknown, but several contributing factors have been identified. These factors include genetic predisposition, environmental stimuli such as stress or sunburn, hormonal changes associated with thyroid disorders or Addison's disease, and the presence of other autoimmune disorders, including type 1 diabetes and rheumatoid arthritis.

Pathophysiology

The pathophysiology of Vitiligo is a complex, multi-factorial, and polygenic disorder, influenced by both genetic and non-genetic factors. Despite extensive research, the precise cause remains unknown. However, two fundamental principles are well-established: the absence of functional melanocytes in Vitiligo-affected skin and the loss of histo-chemically recognized melanocytes due to their destruction.

This destruction is believed to be a gradual process, resulting in a progressive decrease in melanocytes. Several theories attempt to explain the underlying mechanism of Vitiligo. The first theory proposes that nerve endings in the skin release chemicals toxic to melanocytes. The second theory suggests that melanocytes simply self-destruct through apoptosis.

The third and most widely accepted theory is that Vitiligo is an autoimmune disease, where the immune system mistakenly targets and destroys melanocytes. This autoimmune response is thought to be driven by immunological imbalance, including cytokine dysregulation and autoantibody formation.

Classification and Clinical Features

Vitiligo is characterized by well-defined, depigmented patches on the skin, primarily on the face, hands, wrists, and body orifices. Initially, these patches are small but often grow and change shape. Some individuals may experience itching before a new patch appears. The Koebner phenomenon, where new lesions emerge at injury sites, is also common. Vitiligo can significantly impact mental health, with many affected individuals experiencing depression and mood disorders.

Clinically, Vitiligo is classified into two types based on the distribution, extension and number of lesions.

- **Segmental Vitiligo** typically begins in childhood, and spreads rapidly in the affected area. It is unilateral, follows a dermatomal distribution, and does not cross the midline. Often occurring on the face, it involves the hair compartment soon after onset. Segmental Vitiligo can arrest, leaving persistent depigmented patches, and is not associated with autoimmune disorders. Segmental Vitiligo is categorized into three subtypes based on the extent and distribution of depigmentation.
    - **Uni-segmental Vitiligo,** the most common subtype, affects only one dermatome and involves a single, contiguous area of depigmentation.
    - **Bi-segmental Vitiligo** affects two adjacent dermatomes, resulting in two symmetrical or asymmetrical areas of depigmentation.
    - **Multi-segmental Vitiligo** is the most extensive subtype, affecting multiple dermatomes and involving numerous scattered areas of depigmentation.
- **Non-segmental Vitiligo** is characterized by symmetrical depigmentation. It can begin in childhood or later in life, progressing over time. This type involves the hair compartment in later stages and is often linked to personal or family histories of autoimmune disorders. Non-Segmental Vitiligo commonly affects areas sensitive to pressure and friction, such as the hands, fingers, face, and trauma-exposed sites. Non-Segmental Vitiligo is subdivided into Acrofacial, Mucosal, Generalized/Common, Universal, Mixed, and Rare forms.
    - **Acrofacial Vitiligo** affects the face, head, hands, fingers, and feet and preferably involves the perioral region.
    - **Mucosal Vitiligo** targets oral and genital mucosae.
    - **Generalized/Common Vitiligo** presents symmetrically on hands, fingers, face, and trauma-exposed areas.
    - **Universal Vitiligo** covers extensive body surface areas (80-90%), often preceding the generalized form.
    - **Mixed Vitiligo** combines segmental and non-segmental characteristics. Most often, the segmental form precedes non-segmental Vitiligo.
    - **Rare forms of Vitiligo** include Vitiligo punctata, characterized by sharply demarcated, punctiform macules, and Follicular Vitiligo, which primarily affects the follicular reservoir and leads to the whitening of body hairs.. These types were previously considered unclassifiable.

Diagnosis:

Diagnosis is based on clinical features, which are often supplemented by specialized tests. A clinical diagnosis is established through the identification of well-defined, depigmented patches and the confirmed absence of melanocytes in the affected areas.

To affirm the diagnosis, various tests may be employed. The Wood's lamp examination is particularly effective, as it enhances the visibility of depigmented regions, especially in individuals with fair skin. Dermatoscopy facilitates the evaluation of skin morphology and pigment distribution, yielding valuable insights. Although a skin biopsy is rarely required, it serves to confirm the absence of melanocytes. Furthermore, additional tests may be conducted to identify associated conditions. Autoantibody tests are utilized to detect autoimmune disorders, while thyroid function tests are implemented to screen for thyroid abnormalities.

CELLULITIS

Introduction

Cellulitis is an acute bacterial infection that affects the deeper layers of the skin and subcutaneous tissue, typically caused by Streptococci or Staphylococci. Globally, cellulitis affects approximately 1.5% to 3% of the population annually, with incidence rates ranging from 199 to 455 per 100,000 persons per year. In India, the scenario is similar, with an estimated 2.5% to 5% of the population affected annually.

The risk factors for cellulitis are diverse and can be broadly categorized into two groups. The first group includes factors that cause a breakdown in the skin barrier, which can increase the risk of infection. These factors include skin injuries, surgical incisions, intravenous punctures, fissures between toes, insect or animal bites, and other skin infections.

The second group consists of patients with specific co-morbidities that make them more susceptible to developing cellulitis. These conditions include diabetes mellitus, venous insufficiency, peripheral arterial disease, and lymphoedema.

Pathophysiology

The pathophysiology of cellulitis typically begins when bacteria, such as Streptococci or Staphylococci, infiltrate the skin through breaks resulting from injuries, surgical incisions, insect or animal bites, or other skin infections. Once these pathogens penetrate the surface, they invade and colonize the deeper layers of the skin and subcutaneous tissue, thereby eliciting a significant immune response.

In response to the infection, white blood cells are attracted to the affected area, leading to inflammation marked by redness, warmth, and swelling. As bacterial proliferation continues and the immune response escalates, enzymes and toxins produced by the bacteria, in conjunction with inflammatory mediators from immune cells, contribute to tissue damage. This process results in pain, increased swelling, and the potential formation of pus or abscesses. In severe cases, the bacteria may disseminate through the bloodstream, resulting in systemic manifestations such as fever, chills, and malaise. If left untreated, cellulitis can progress to septicemia, a life-threatening condition.

Clinical features:

Cellulitis is characterized by distinct local signs and symptoms, including erythema, oedema, warmth, tenderness, pain, and induration (hardening of the skin). The affected area may present with additional features such as skin lesions or wounds, ulcerations, abscesses, or fistulas. Furthermore, certain cases of cellulitis may exhibit vesicles or bullae (blister-like structures).

In severe cellulitis, systemic signs and symptoms may occur such as, fever, chills, malaise, fatigue, headache, nausea, vomiting, and Lymphadenopathy. The severity of cellulitis can be classified into three categories: mild, moderate, and severe.

- **Mild cases** are localized with minimal systemic symptoms,
- **Moderate cases** are widespread with moderate systemic symptoms.
- **Severe cases** are extensive, with severe systemic symptoms and potentially life-threatening complications.

Untreated or severe cellulitis can lead to complications such as abscess formation, gangrene, necrotizing fasciitis, sepsis, septicemia, and organ failure. Special considerations include facial cellulitis, which may lead to orbital cellulitis or cavernous sinus thrombosis, and Periorbital cellulitis, which may result in vision loss.

Diagnosis:

Diagnosis is based on clinical evaluation, blood cultures and tissue cultures. A clinical diagnosis primarily depends on the presence of spreading erythematous inflammation in the deep dermis and subcutaneous tissue. Characteristic symptoms encompass worsening erythema, oedema, warmth, and tenderness. A diagnosis requires the fulfillment of at least two of the following four criteria: warmth, erythema, oedema, or tenderness.

Blood cultures are useful in detecting or ruling out bacteremia, particularly in immuno-compromised patients, and in assessing individuals who exhibit signs of systemic infection, uch as fever and leukocytosis. Tissue cultures may be indicated for immuno-compromised **atients** who do not respond to empirical treatment, as well as for cases of cellulitis associated *ith specific* injuries, including animal bites and penetrating wounds.

MCQ's

1. What is the most common form of eczema in infants

a) Seborrhoeic dermatitis b) Atopic dermatitis

c) Nummular eczema d) Stasis dermatitis

2. Which gene mutation is frequently associated with eczema

a) FLG gene b) HLA gene

c) BRCA1 gene d) CFTR gene

3. Which type of eczema is associated with fluid-filled blisters on the palms and soles

a) Atopic dermatitis b) Seborrhoeic dermatitis

c) Pompholyx d) Asteatotic eczema

4. Which type of eczema is induced by poor circulation in the legs

a) Asteatotic eczema b) Stasis dermatitis

c) Juvenile plantar dermatitis d) Infectious eczemoid dermatitis

5. Which type of urticaria is associated with autoimmune disorders

a) Acute urticaria b) Chronic urticaria

c) Inducible urticaria d) Spontaneous urticaria

6. What is a common characteristic of dermatographism in urticaria

a) Wheals triggered by minor trauma b) Persistent itching

c) Presence of angioedema d) Recurring wheals

7. Which type of psoriasis is characterized by smooth, erythematous, and well-defined patches in intertriginous areas

a) Plaque psoriasis b) Inverse psoriasis

c) Guttate psoriasis d) Pustular psoriasis

8. What is the hallmark sign of Auspitz in psoriasis

a) Thickened nail plates

b) Small, non-infectious pustules

c) Multiple bleeding points upon skin scraping

d) Raindrop-shaped plaques

9. What are the characteristic lesions of Lichen planus
   - a) Large red blisters
   - b) Small, shiny, violaceous, flat-topped papules
   - c) Circular patches with clear centers
   - d) Fluid-filled vesicles

10. Which of the following is NOT a common site for Lichen planus lesions
    - a) Wrists b) Scalp
    - c) Trunk d) Medial thighs

11. What does a skin or mucous membrane biopsy help to exclude in the diagnosis of Lichen planus
    - a) Lichenoid dermatitis b) Psoriasis
    - c) Discoid lupus erythematosus d) All of the above

12. What are desmogleins
    - a) Hormones in the immune system b) Adhesion molecules in the skin
    - c) Genetic markers d) Antibiotics

13. What is the most prevalent form of pemphigus
    - a) Pemphigus Foliaceus b) Pemphigus Erythematosus
    - c) Paraneoplastic Pemphigus d) Pemphigus Vulgaris

14. What is the key histopathological feature of pemphigus
    - a) Hyperkeratosis b) Acantholysis
    - c) Granuloma formation d) Dermal fibrosis

15. What characterizes Mucous Membrane Pemphigoid
    - a) Large, tense blisters on the skin
    - b) Erosions, ulcers, and scarring in mucous membranes
    - c) Blisters triggered by medications
    - d) Intense pruritus around the umbilicus

16. What is a common causative agent of superficial mycoses
    - a) Candida albicans b) Histoplasma capsulatum
    - c) Aspergillus d) Sporothrix

17. Which type of mycoses primarily affects the subcutaneous tissues
    - a) Dermatophytosis b) Onychomycosis
    - c) Sporotrichosis d) Cutaneous aspergillosis

18. What are the characteristic clinical features of tinea pedis
    - A) Itching and burning sensations, cracked skin between toes
    - B) Circular rashes with clear centers
    - C) Pustules and redness on the face
    - D) Skin and nail discoloration

19. What is the primary causative agent of leprosy
    - a) Mycobacterium tuberculosis b) Mycobacterium leprae
    - c) Mycobacterium avium d) Mycobacterium kansasii

20. What is the typical incubation period of leprosy
    - a) 1 month to 1 year b) 3 to 5 years
    - c) 10 to 20 years d) 1 to 10 days

21. Which type of leprosy is characterized by a higher bacterial load and more severe symptoms
    - a) Paucibacillary Leprosy (PB) b) Multibacillary Leprosy (MB)
    - c) Tuberculoid Leprosy (TT) d) Borderline Tuberculoid Leprosy (BT)

22. What is the primary immune response in tuberculoid leprosy
    - a) Strong cell-mediated immunity b) Strong humoral immunity
    - c) Weak cell-mediated immunity d) Weak humoral immunity

23. Which diagnostic method is used to visualize Mycobacterium leprae in skin smears
    - a) ELISA b) Ziehl-Neelsen stain
    - c) PCR d) Immunofluorescence

24. What is the Koebner phenomenon associated with Vitiligo
    - a) Appearance of new lesions at sites of injury
    - b) Gradual darkening of the skin
    - c) Spontaneous disappearance of white patches
    - d) Emergence of painful blisters

25. Which form of Vitiligo is characterized by a dermatomal distribution and does not cross the midline

a) Non-segmental Vitiligo b) Acrofacial Vitiligo

c) Segmental Vitiligo d) Universal Vitiligo

26. What test enhances the visibility of depigmented regions, especially in fair-skinned individuals

a) Dermatoscopy b) Wood's lamp examination

c) Biopsy d) PCR test

27. Which type of vitiligo affects oral and genital mucosae

a) Acrofacial Vitiligo b) Mucosal Vitiligo

c) Generalized Vitiligo d) Segmental Vitiligo

28. What is the primary causative agent of cellulitis

a) Candida albicans b) Streptococci or Staphylococci

c) Mycobacterium leprae d) Histoplasma capsulatum

29. What are the characteristic local signs of cellulitis

a) Vesicles and pustules

b) Erythema, oedema, warmth, tenderness, pain, and induration

c) Hair loss and pitting

d) Itchy, red, scaly patches

30. Which diagnostic method is used to detect bacteremia in cellulitis patients

a) Skin prick test

b) Wood's lamp examination

c) Blood cultures

d) ELISA

31. What are potential complications of untreated or severe cellulitis

a) Gangrene, sepsis, septicemia, and organ failure

b) Melanoma

c) Hyperpigmentation

d) Cold sores

Answer Keys

1	b	2	a	3	c	4	b	5	b	6	a	7	b	8	c	9	b	10	b
11	d	12	b	13	d	14	b	15	b	16	a	17	c	18	a	19	b	20	b
21	b	22	a	23	b	24	a	25	c	26	b	27	b	28	b	29	b	30	c
31	a																		

Short Answer Questions

1. Explain Eczema
2. Explain Urticaria
3. Explain Psoriasis
4. Explain Lichen Planus
5. Write the difference between Pemphigus and Pemphigoid
6. Explain Leprosy
7. Explain Vitiligo
8. Explain Cellulitis

◆

TOPIC 34

Galagaṇḍa, Gaṇḍamālā and Apacī

Introduction

Galagaṇḍa, Gaṇḍamālā and Apacī are the diseases occurring mainly in the Gala pradeśa (neck region) with the vitiation of Medas and Kapha. In general, Galagaṇḍa is a solitary swelling manifesting in the Galapārśva (side of the neck), whereas Gaṇḍamālā is Bahubhistu Gaṇḍa (multiple glandular swelling) in the neck which looks like garland and Apacī is suppurated glandular swelling manifesting in the Hanvasthi (mandible), Kakṣa (axilla), Akṣaka (clavicle), Bāhusandhi (shoulder joint), Manyā (nape of the neck) and Gala pradeśa (neck region). There are difference of opinion among Ācāryās regarding Gaṇḍamālā and Apacī.

Ācārya Caraka described Gaṇḍamālā not Apacī; whereas Ācārya Suśruta explained Apacī not Gaṇḍamālā; and Ācārya Vāgbhaṭa described Gaṇḍamālā and Apacī together with similar symptoms. Ācārya Bhoja opines that Gaṇḍamālā is the type of Apacī which starts to manifest in the form of garland in the region of mandible, neck and heart. Ācārya Madhukośa opines the same that Amālārūpa (not in garland form) is Apacī. Ācārya Cakrapāṇi opines that Granthi Visarpa is Apacī.

Nirūkti and Paribhāṣa of Galagaṇḍa, Gaṇḍamālā and Apacī

Galagaṇḍa

- गले गण्डः स्फोटक इव । (SKD)

That which causes the Gaṇḍa (swelling or boil) in the Gala pradeśa (neck region) similar to Sphoṭaka (tumour) is known as Galagaṇḍa.

- निबद्धः श्वयथुर्यस्य मुष्कवल्लम्बते गले । महान् वा यदि वा ह्रस्वो गलगण्डं तमादिशेत् ॥ (Su.Ni.11/29)

That which causes Mahān (big) or Hrasva Śvayathu (small swelling) and it hangs like Muṣka (scrotum) in the Gala pradeśa (neck region) is known as Galagaṇḍa.

- महान्तं शोथमल्पं वा हनुमन्यागलाश्रयम्। लम्बन्तं मुष्कवद्दृष्ट्वा गलगण्डं विनिर्दिशेत्। ॥ (Ma.M.N.38/1)

That which causes the Mahāntaṃ (big) or Alpa Śotha (small swelling) and it hangs like Muṣka (scrotum) in the Hanumanyāgala pradeśa (chin, nape of neck and neck region) is known as Galagaṇḍa.

- पवनश्लेष्ममेदोभिर्गलगण्डो भवेद्बहिः। वर्धमानः स कालेन मुष्कवल्लम्बतेऽतिरुक्॥ (A.H.U.21/53)

That which causes Gaṇḍa (swelling) in the Bahiḥgala (outside of the throat) due to aggravation of Vāta, Kapha and Medas and is characterized by Vardhamānaḥ sa kālena (growing in size in course of time), Muskavallambate (hangs like scrotum) and Atiruk (severe pain) is known as Galagaṇḍa.

- गलस्य पार्श्वे गलगण्ड एकः स्याद्गण्डमाला बहुभिस्तु गण्डैः। (Ca.Ci.12/79)

That which causes Eka Gaṇḍa (single swelling) in the Gala pārśva (sides of the neck) is known as Galagaṇḍa and Gaṇḍamālā is Bahubhistu gaṇḍa (multiple glandular swelling).

Gaṇḍamālā

- गण्डानां ग्रीवाजातस्फोट विशेषाणां माला समूहोऽस्याम् । (SKD)

That which causes the Gaṇḍa (swelling) in the Gala pradeśa (neck region) similar to Sphoṭa (tumour) in the garland form is known as Gaṇḍamālā.

- स्याद्गण्डमालेति मालातुल्यगण्डयोगात् गण्डमाला। (Ma.M.N.38/11)
- गले गण्डकरूपा व्रणा एवं गंण्डमालादयो ज्ञेयाः । मालातुल्यं गण्डयोगात् गण्डमाला ॥ (Āḍ.Śā.P.7/67)

When the Gaṇḍaka (swelling) in the Gala pradeśa (neck region) resembles the Vraṇā (tumour) and appears alike to Mālā (garland) it is known as Gaṇḍamālā.

- कर्कन्धुकोलामलकप्रमाणैः कक्षांसमन्यागलवङ्क्षणेषु ॥ मेदःकफाभ्यां चिरमन्दपाकैः स्याद्गण्डमाला बहुभिश्च गण्डैः । (M.N.38/8-9)

Due to aggravation of Medas and Kapha doṣa, there occurs Bahubhiśca Gaṇḍa (multiple swelling) in the Kakṣāṃsamanyāgalavaṃkṣaṇa (axilla, shoulder, nape of the neck, neck and groin region) resembling the Karkandhu, Kola and Āmalaka pramāṇa (size of Karkandhu, Kolaphala and Āmalaka phala) and it takes long period for suppuration; it is known as Gaṇḍamālā.

- गण्डमालिका कण्ठदेशे मालाकारा ग्रन्थिरूपा साप्येकप्रकार अपचीभेदः ॥ (Kā.Śā.P.7/67)

Granthi (knot or tumor) in the Kaṇṭha pradeśa (neck region) similar to Mālākāra (garland form) is known as Gaṇḍamālā and it is a type of Apacī.

Apacī

- तमुपचीयमानं खलु अन्यैर्ग्रन्थिभिरामलकास्थिप्रमाणैर्मीनाण्डाकारैः। (Ga.Su.Ni.11/10-12)

Granthi (knot or tumor) in a size of Āmalakāsthi (kernel of a gooseberry) and resembling the Aṇḍākāra (shape of egg) is known as Apacī.

- ⊙ चयप्रकर्षादपचीमिति निरुक्तिः । (Ḍa.Su.Ni.11/10-12)

Granthi (knot or tumor) which is characterized by Cayaprakarṣāt (intensely increasing or continuous growth) is known as Apacī.

- ⊙ सैवापचीति तादृशी अमालारूपा अपची भण्यते। (Ma.M.N.38/9-10)

That which causes Granthi (knot or tumor) in such a manner like that in Amālārūpa (not in garland form) is known as Apacī.

Saṃprāpti of Galagaṇḍa, Gaṇḍamālā and Apacī

Galagaṇḍa

- ⊙ वातः कफश्चैव गले प्रवृद्धौ मन्ये तु संसृत्य तथैव मेदः । कुर्वन्ति गण्डं क्रमशः स्वलिङ्गैः समन्वितं तं गलगण्डमाहुः ॥ (Su.Ni.11/22)

The aggravated Vāta and Kapha being accumulated in the Gala (neck) and Manya (nape of the neck) along with the Medas produces Gaṇḍa (swelling) with its own clinical features; it is called Galagaṇḍa.

Gaṇḍamālā and Apacī

- ⊙ हन्वस्थिकक्षाक्षकवाहुसन्धिमन्यागलेषूपचितं तु मेदः । ग्रन्थिं स्थिरं वृत्तमथायतं वा स्निग्धं कफश्चाल्परुजं करोति ॥ तं ग्रन्थिभिस्त्वामलकास्थिमात्रैर्मत्स्याण्डजालप्रतिमैस्तथाऽन्यैः। अनन्यवर्णैरुपचीयमानं चयप्रकर्षादपचीं वदन्ति ॥ कण्डूयुतास्तेऽल्परुजः प्रभिन्नाः स्रवन्ति नश्यन्ति भवन्ति चान्ये । मेदःकफाभ्यां खलु रोग एष सुदुस्तरो वर्षगणानुबन्धी ॥ (Su.Ni.11/10-12)

The aggravated Medas and Kapha gets accumulated in the Hanvasthi (mandible), Kakṣa (axilla), Akṣaka (clavicle), Bāhusandhi (shoulder joint), Manyā (nape of the neck) and Gala pradeśa (neck region) produces Granthi (knot or tumor) which are Sthira (fixed), Vṛtta (round in shape), Snigdha (unctuous) associated with Mandaruja (mild pain). Some of these Granthis resembles Āmalakāsthi mātra (kernel of a gooseberry in size) or Matsyāṇḍa (the mass of fish eggs) and having the Ananyavarṇairupa (same colour of the skin). Due to Cayaprakarṣāt (continuous growth or intensely increasing), it is known as Apacī. This is associated with Kaṇḍu (itching), Alparuja (mild pain), Prabhinna sravanti naśyanti bhavanti cānye (when burst they discharge and disappear, while other appears). Thus this disease is caused by Medas and Kapha and is Dustara (difficult to treat) and Varṣagaṇānubandhī (persists for several years).

- ⊙ मेदस्थाः कण्ठमन्याक्षकक्षावङ्क्षणगा मलाः। सवर्णान् कठिनान् स्निग्धान् वार्ताकामलकाकृतीन्॥ अवगाढान् बहून् गण्डांश्चिरपाकांश्च कुर्वते। पच्यन्तेऽल्परुजस्तेऽन्ये स्रवन्त्यन्येऽतिकण्डुराः॥ नश्यन्त्यन्ये भवन्त्यन्ये दीर्घकालानुबन्धिनः। गण्डमालाऽपची चेयं दूर्वेव क्षयवृद्धिभाक्। (A.H.U.29/23-25)

The aggravated doṣas afflicts the Medas in the Kaṇṭhamanyākṣavakṣāvaṃkṣaṇā (throat, neck, clavicle region, axilla and the groin regions) and produces the Gaṇḍa (swelling) which is Savarṇān (of same colour of the skin), Kaṭhinān (hard), Snigdhān (unctuous), Vārtākāmalakākṛti (resembles the Vārtaka and Āmalaka in size), Avagāḍha (deep rooted), Bahūn (multiple), and Cirapāka (undergoes suppuration after a long time). Anye pacyantealparuja (some undergoes suppuration causing mild pain), Sravanyanyeatikaṇḍurā (others exude fluid causing severe itching), Naśyantyane bhavantyanye (some disappears and some appears fresh) and Dīrghakālanubandhini (it persists for long time). This disease is known as Gaṇḍamālā or Apacī which undergoes Dūrveva kṣayavṛddhibhāk (decrease and increase like Dūrvā grass).

- ⊙ वातपित्तकफा वृद्धा मेदश्चापि समाचितम्। जङ्घयोः कण्डराः प्राप्य मत्स्याण्डसदृशान् बहून्॥ कुर्वन्ति ग्रथितांस्तेभ्यः पुनः प्रकुपितोऽनिलः। तान् दोषानूर्ध्वगो वक्षःकक्षामन्यागलाश्रितः॥ नानाप्रकारान् कुरुते ग्रन्थीन् सा त्वपची स्मृता। व्यामिश्रदोषजाता सा कृच्छ्रसाध्या प्रकीर्तिता। (Ḍa.Su.Ni.11/10-12)

Ācārya Bhoja described that the aggravated Vāta, Pitta, Kapha and Medas reach the Jaṅghayoḥ kaṇḍarā (vessels in the calf region) produces Bahūn granthi (multiple tumors) which resembles the Matsyāṇḍa (fish eggs). Further the Vāta gets aggravated and moves upwards and produces Nānaprakāra granthi (different types of tumors) in the Vakṣaḥkakṣāmanyāgala pradeśa (chest, axilla, nape of the neck and neck region) resulting in the disease Apacī and it is Kṛcchrasādhyā (difficult to treat) due to Vyamiśradoṣajāta (mixed doṣas together).

Saṃprāpti Ghaṭaka of Galagaṇḍa, Gaṇḍamālā and Apacī

Saṃprāpti Ghaṭaka	Galagaṇḍa	Gaṇḍamālā and Apacī
Doṣa	Vāta and Kapha	Kapha Pradhāna Tridoṣas
Dūṣya	Medas	Medas
Agni	Jaṭharāgni māndhya	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma	Jaṭharāgni māndhya janya Āma
Srotas	Medovaha	Medovaha
Srotoduṣṭi	Saṅga	Saṅga, Atipravṛtti
Sañcāra sthāna	Gala	Gala
Adhiṣṭhāna	Rohiṇī (6th layer of Tvak)	Rohiṇī (6th layer of Tvak)
Udbhava sthāna	Āmapakvāśaya	Āmapakvāśaya
Vyakta sthāna	Gala pradeśa	Hanvasthikakṣākṣakanāhusandhi manyā gala pradeśa
Rogamārga	Bāhya	Bāhya

Bheda and Lakṣaṇās of Galagaṇḍa, Gaṇḍamālā and Apacī

Galagaṇḍa

Ācārya Suśruta classified Galagaṇḍa into three types namely Vātaja, Kaphaja and Medoja Galagaṇḍa.

Vātaja Galagaṇḍa

- तोदान्वितः कृष्णसिरावनद्धः कृष्णोऽरुणो वा पवनात्मकस्तु । मेदोन्वितश्चोपचितश्च कालाद्भवेदतिस्निग्धतरोऽरुजश्च । पारुष्ययुक्तश्चिरवृद्ध्यपाको यदृच्छया पाकमियात् कदाचित् वैरस्यमास्यस्य च तस्य जन्तोर्भवेत्तथा तालुगलप्रशोषः ॥ (Su.Ni.11/23-24)
- कृष्णोऽरुणो वा तोदाढ्यः स वातात्कृष्णराजिमान्। वृद्धस्तालुगले शोषं कुर्याच्च विरसास्यताम्। (A.H.U.21/54)

The clinical features of Vātaja Galagaṇḍa are Kṛṣṇoaruṇo vā (black or reddish colored swelling) associated with Todānvitaḥ (pricking pain inside), Kṛṣṇāsirāvanaddha (appearance of blackish veins on its surface). When it is associated with Medas in due course of time, it becomes Upacita (increase in size), Atisnigdha (excessive unctuousness) and Aruja (painless). And the Granthi is Pāruṣyayukta (rough to touch), Ciravṛddha (gradually increasing in size), Apākoyadṛcchayā pākamityāt (not suppurates or rarely suppurates), Āsyavairasya (loss of taste in the mouth) and Tālugalapraśoṣa (dryness in the throat and the palate).

Kaphaja Galagaṇḍa

- स्थिरः सवर्णोऽल्परुगुग्रकण्डूः शीतो महांश्चापि कफात्मकस्तु । चिराभिवृद्धिं कुरुते चिराच्च प्रपच्यते मन्दरुजः कदाचित् ॥ माधुर्यमास्यस्य च तस्य जन्तोर्भवेत्तथा तालुगलप्रलेपः । (Su.Ni.11/25-26)
- स्थिरः सवर्णः कण्डूमान् शीतस्पर्शो गुरुः कफात्। वृद्धस्तालुगले लेपं कुर्याच्च मधुरास्यताम्॥ (A.H.U.21/55)

The clinical features of Kaphaja Galagaṇḍa are Sthira (firm), Savarṇa (same colour of skin), Alparuja (mild pain), Śīta (cold), and Mahān (big in shape). Cirābhivṛddhiṃ (it increase in size after a long period), Cirācca Prapacyate (rarely suppurates), Mandaruja (mild pain), Mādhuryāsyata (sweet taste in the mouth) and Tālugalapralepa (feeling as if the throat and palate is smeared)

Medoja Galagaṇḍa

- स्निग्धो मृदुः पाण्डुरनिष्टगन्धो मेदःकृतो नीरुगथातिकण्डूः ॥ प्रलम्बतेऽलाबुवदल्पमूलो देहानुरूपक्षयवृद्धियुक्तः । स्निग्धास्यता तस्य भवेच्च जन्तोर्गलेऽनुशब्दं कुरुते च नित्यम् ॥ (Su.Ni.11/26-27)
- मेदसः श्लेष्मवद्धानिवृद्ध्योः सोऽनुविधीयते। देहं वृद्धश्च कुरुते गले शब्दं स्वरेऽल्पताम्॥ (A.H.U.21/56)

The clinical features of Medoja Galagaṇḍa are Snigdha (smooth or unctuous), Mṛdu (soft), Pāṇḍu (pale colored), Aniṣṭagandha (unpleasant odour), Niruk (painless), Atikaṇḍu (excessive itching). It hangs like Alābuvadalpamūlo (Alābu with a narrow base), Dehānurūpakṣayavṛddhiyukta (increase and decrease in size occurs corresponding to the body), Snigdhāsyata (stickiness of the mouth) and Nityam Galeanuśabdaṃ (constant sound heard in the throat).

Gaṇḍamālā and Apacī

Ācārya Bhoja classified Apacī into four types namely Vātaja, Pittaja, Kaphaja and Medoja Apacī.

- तासां वातोत्तरा रूक्षा वातवेदनयाऽन्विता। क्षिप्रपाकसमुत्थाना दाहयुक्ता तु पैत्तिकी॥ गूढा वाऽपाककठिना कफात् स्निग्धाऽल्परुक्करा। मेदोऽधिका श्लैष्मिकी च विशेषादतिमार्दवा॥ (Ḍa. Su.Ni.11/11-12)

- **Vātaja Apacī:** It is characterized by Rūkṣā (non-unctuousness) accompanied with Vātavedana (different types of Vātaja type of pain)
- **Pittaja Apacī:** It is characterized by Dāha (burning sensation) along with Kṣiprapāka (suppurates quickly)
- **Kaphaja Apacī:** It is characterized by Gūḍha (concealed swelling) or Apāka (not suppurates), Kaṭhina (hard), Snigdha (smooth or unctuous) and Alpa ruk (mild pain).
- **Medoja Apacī:** It is similar to Kaphaja Apacī features especially with Atimārdava (excessive softness)

Sādhyāsādhyata of Galagaṇḍa, Gaṇḍamālā and Apacī

- कृच्छ्राच्छ्वसन्तं मृदुसर्वगात्रं संवत्सरातीतमरोचकार्तम् । क्षीणं च वैद्यो गलगण्डिनं तु भिन्नस्वरं चैव विवर्जयेत्तु ॥ (Su.Ni.11/28)

In case of Galagaṇḍa, if the patients suffers from Kṛcchācchvasanta (difficulty in respiration), Mṛdusarvagātra (the whole body becomes soft), Saṃvatsara (more than a year), Atitamarocaka (excessive anorexia), Kṣīṇa (emaciation) and Bhinnasvara (hoarseness of voice), then the physician should be discard the patient from the treatment as it is incurable.

- साध्याः स्मृताः पीनसपार्श्वशूलकासज्वरच्छर्दियुतास्त्वसाध्याः॥ (Ca.Ci.12/79)

Gaṇḍamālā and Galagaṇḍa are Sādhya (curable), however it becomes Asādhya (incurable), if it is associated with Pīnasa (nasal catarrah), Pārśvaśūla (pain in the flanks), Kāsa (cough), Jvara (fever) and Chardi (vomiting)

- तां त्यजेत्सज्वरच्छर्दिपार्श्वरुक्कासपीनसाम्। (A.H.U.29/26)

Gaṇḍamālā and Apacī should be refused from the treatment if it is associated with Jvara (fever), Chardi (vomiting), Pārśvaruk (pain in the flanks), Kāsa (cough) and Pīnasa (nasal catarrah).

In Contemporary science,

Galagaṇḍa, Gaṇḍamālā and Apacī cannot be correlated with a single disease in contemporary science. Any swelling around the neck can be considered as Galagaṇḍa, Gaṇḍamālā and Apacī. Goiter, hypothyroidism, cervical lymphadenitis, etc., are the common diseases correlated with Galagaṇḍa, Gaṇḍamālā and Apacī. Some scholars opine that Galagaṇḍa can be correlated with goiter and hypothyroidism. Gaṇḍamālā and Apacī can be correlated with stages of cervical lymphadenitis. Some others opine that diffuse, nodular or multinodular goiter can be correlated with Galagaṇḍa, Gaṇḍamālā and Apacī.

MCQ's

1. According to Caraka, Granthi Visarpa is
   a) Galagaṇḍa
   b) Gaṇḍamālā
   c) Apacī
   d) Granthi
2. Galagaṇḍa is
   a) Eka Gaṇḍa in Gala Pārśva
   b) Bahubhistu gaṇḍa
   c) Āmalakāsthi pramāṇa Granthi
   d) None of the above
3. According to Mādhavakara, Apacī is
   a) Mālārūpa
   b) Granthi Visarpa
   c) Amālārūpa
   d) All of the above
4. मुष्कवल्लम्बते is the feature of
   a) Galagaṇḍa
   b) Gaṇḍamālā
   c) Apacī
   d) Granthi
5. मालाकारा ग्रन्थिरूपा is the feature of
   a) Galagaṇḍa
   b) Gaṇḍamālā
   c) Apacī
   d) Granthi
6. Doṣa Predominance in Galagaṇḍa
   a) Vātakapha
   b) Vātapitta
   c) Kaphapitta
   d) Sannipātaja

7. According to Suśruta, Galagaṇḍa is of .... types
   a) 3
   b) 4
   c) 5
   d) 6
8. According to Bhoja, Apacī is of _ types
   a) 3
   b) 4
   c) 5
   d) 6
9. प्रलम्बतेऽलाबुवदल्पमूल is the feature of
   a) Vātaja Galagaṇḍa
   b) Kaphaja Galagaṇḍa
   c) Medoja Galagaṇḍa
   d) None of the above
10. देहानुरूपक्षयवृद्धियुक्तः is the feature of
    a) Vātaja Galagaṇḍa
    b) Kaphaja Galagaṇḍa
    c) Medoja Galagaṇḍa
    d) Medoja Apacī

Answer Keys

1	c	2	a	3	c	4	a	5	b	6	a	7	a	8	b	9	c	10	c

◆

TOPIC 35

Thyroid Disorders

Introduction

Thyroid disorders comprise a spectrum of conditions affecting the thyroid gland, a small endocrine gland with a butterfly-shaped structure situated in the anterior neck just below the cricoid cartilage, consisting of two lobes connected by an isthmus. This gland plays a crucial role in regulating metabolism and overall bodily functions through the synthesis and secretion of thyroid hormones, including inactive thyroid hormone (Tetraiodothyronine or thyroxine or T4) and active thyroid hormone (Triiodothyronine or T3). Additionally, the parafollicular cells (C cells) secrete calcitonin, contributing to the regulation of blood calcium levels. Imbalances in thyroid function can lead to a diverse range of health complications, contingent upon whether the gland is hyperactive (hyperthyroidism) or underactive (hypothyroidism).

HYPERTHYROIDISM

Introduction

Hyperthyroidism is a medical condition characterized by the excessive production of thyroid hormones, leading to thyrotoxicosis. This is caused by increased synthesis and secretion of thyroid hormones due to thyroid-stimulating immunoglobulin (TSI), autonomous thyroid hyperfunction, or excessive thyroid hormone release without increased synthesis. The global prevalence of hyperthyroidism varies and is influenced by dietary iodine content. Women are more affected than men, with a female-to-male ratio ranging from 5:1 to 10:1. Additional risk factors include smoking, iodine deficiency or excess, selenium deficiency, genetic predisposition, and the use of certain medications such as lithium and amiodarone.

The most common causes of hyperthyroidism are Graves' disease (diffuse toxic goitre), toxic multinodular goitre, and a toxic adenoma. Less frequent causes include hyper secretion of pituitary thyroid-stimulating hormone (TSH) by a pituitary tumor, hyper secretion of thyrotropin-releasing hormone (TRH), thyroiditis, metastatic tumors of the thyroid, struma ovarii, congenital hyperthyroidism in the newborn of a mother with Graves' disease, hCG-secreting tumors due to mild thyrotropic effects of hCG (e.g. hydatidiform mole, choriocarcinoma, and testicular tumors), and excessive doses of thyroid hormones or iodine, known as Jod-Basedow disease..

Pathophysiology:

The secretion and release of thyroid hormones are meticulously regulated by a complex negative feedback loop involving the hypothalamus, pituitary gland, and thyroid gland. The hypothalamus releases TRH (thyrotropin-releasing hormone), which stimulates the pituitary to release TSH (thyroid-stimulating hormone), in turn stimulating the thyroid gland to release T4 and T3. The increased thyroid hormone synthesis typically causes inhibition of TRH and TSH release by the hypothalamus and pituitary, respectively. Disruption of this intricate system can result in hyperthyroidism, with TSH-secreting pituitary tumours posing a particular challenge due to their release of hormones unresponsive to normal feedback control, potentially causing symptoms such as amenorrhea, galactorrhea, or signs of acromegaly. Additionally, thyrotoxicosis can occur when autonomous follicles produce excess thyroid hormone. The synthesis of thyroid hormone in the thyroid gland depends on iodine, and any process that increases the level of unbound (free) thyroid hormone can induce thyrotoxicosis.

Clinical Features:

The clinical features of hyperthyroidism include nervousness, anxiety, palpitations, hyperactivity, increased sweating, heat intolerance, fatigue, increased appetite, weight loss, insomnia, weakness, and frequent bowel movements (occasionally diarrhea). Hypomenorrhea may also be present. Physical signs can include warm, moist skin, tremor, tachycardia, widened pulse pressure, onycholysis, lidlag (refers to a condition where the upper eyelid is positioned higher than normal when the eye is looking downward) and atrial fibrillation. In men, gynecomastia may sometimes occur.

Complications:

Untreated hyperthyroidism may progress to a severe condition termed thyroid storm, characterized by the hypermetabolic state typical of hyperthyroidism. Patients in thyroid storm typically manifest tachycardia, heightened gastrointestinal motility, diaphoresis, anxiety, fever, and signs of multi-organ dysfunction. Prolonged untreated hyperthyroidism is correlated with an increased risk of acute cardiovascular events, atrial fibrillation, ischemic stroke, osteoporosis, infertility, menstrual irregularities, and increased mortality. Furthermore, subclinical hyperthyroidism has been linked to an augmented risk of arrhythmias such as atrial fibrillation, osteoporosis, hip fractures, and increased mortality.

Diagnosis:

Diagnosis of hyperthyroidism is based on clinical examination, and thyroid profile tests. Clinical findings may include tachycardia (with some patients exhibiting atrial fibrillation), hypertension, tremors, warm and moist skin, hyperreflexia, and an anxious countenance. Additionally, signs of heart failure may be present. Thyroid examination may reveal a diffuse, non-nodular enlargement in Graves' disease, a diffuse, non-symmetric nodular enlargement in toxic multinodular goiter and a single large nodule in cases of a toxic adenoma. Notably, subacute thyroiditis may present with an exquisitely tender thyroid.

Thyroid profile tests typically demonstrate low or suppressed TSH levels alongside elevated free T4 and total T3 levels in overt hyperthyroidism. T3 toxicosis, a specific subtype, is characterized by elevated T3 levels accompanied by low or suppressed TSH and normal T4 levels. Subclinical hyperthyroidism is indicated by low or suppressed TSH with normal T3 and T4 levels.

Upon confirming the diagnosis of hyperthyroidism, thyrotropin receptor antibody (TRAb) levels are measured as an initial test to ascertain the aetiology of hyperthyroidism. Elevated TRAb levels confirm the diagnosis of Graves' disease.

HYPOTHYROIDISM

Introduction

Hypothyroidism is an endocrine disorder characterized by insufficient production of thyroid hormones, which are crucial for regulating metabolism, growth, and development. This condition affects approximately 5-10% of the global population, with a higher prevalence among women (6-12%) than men (3-6%). In India, the prevalence of hypothyroidism is notably elevated, ranging from 11.2% in urban areas to 15.4% in rural areas. Subclinical hypothyroidism, a less severe form, affects 10.3% (urban) to 14.5% (rural) of the Indian population. Factors contributing to the high prevalence of hypothyroidism in India include female gender, age over 50, pregnancy women, family history, autoimmune disorders, iodine imbalance, radiation exposure, thyroid surgery, and certain medications.

Hypothyroidism is primarily categorized as primary and secondary hypothyroidism. Primary hypothyroidism results from inadequate function of the thyroid gland. The most prevalent cause of primary hypothyroidism is iodine deficiency in iodine-deficient geographic areas worldwide. In regions with sufficient iodine levels, the leading cause is the autoimmune disease Hashimoto's thyroiditis. Other causes of primary hypothyroidism include postpartum thyroiditis, subacute granulomatous thyroiditis (Quervain's disease), thyroid surgery, radiotherapy of the head or neck area, and certain medications such as **amiodarone**, thalidomide, and oral tyrosine kinase inhibitors.

Secondary and tertiary hypothyroidism, also known as central hypothyroidism, is attributed to a defect in the hypothalamic-pituitary axis secondary to neoplastic, infiltrative, inflammatory, genetic, or iatrogenic disorders of the pituitary or hypothalamus, pituitary tumours, tumours compressing the hypothalamus, Sheehan syndrome, thyroid-releasing hormone (TRH) resistance, TRH deficiency, lymphocytic hypophysitis, radiation therapy to the brain, and medications such as dopamine, prednisone, or opioids.

Subclinical hypothyroidism is often observed in patients with underlying Hashimoto's thyroiditis. Other causes include post-surgical or post-ablative hypothyroidism, central hypothyroidism, and medication-induced hypothyroidism such as lithium, amiodarone, checkpoint inhibitors, and tyrosine kinase inhibitors.

Pathophysiology:

Hypothyroidism is commonly caused by inadequate production of thyroid hormone by the thyroid gland, and less commonly by pituitary and hypothalamus dysfunction. The hypothalamus secretes thyrotropin-releasing hormone (TRH), stimulating the pituitary gland to produce thyroid-stimulating hormone (TSH), which in turn stimulates the thyroid gland to produce mainly T4 and smaller amounts of T3. Any disruption in these processes can lead to hypothyroidism. Additionally, reduced T4 production increases TSH secretion, resulting in hypertrophy and hyperplasia of the thyroid parenchyma and increased T3 production.

Clinical Features:

Hypothyroidism is often asymptomatic, but common symptoms include fluid retention, puffiness around the eyes, fatigue, cold intolerance, weight gain, tingling or numbness in the hands and feet, facial puffiness, sparse, coarse, and dry hair, rough, dry, scaly, and thick skin, carotenemia (particularly evident on the palms and soles due to the accumulation of carotene in the lipid-rich epidermal layers), and macroglossia, constipation, heavy menstrual bleeding or secondary amenorrhea, hoarse voice, and slow speech, depression, and cognitive impairment. In rare cases, patients may present with ascites, rhabdomyolysis, and pericardial effusion.

Complications:

Untreated hypothyroidism may contribute to a range of health complications, including atherosclerosis, heightened risk of heart failure and stroke, hypertension, cardiac arrhythmias, cognitive impairment such as memory and concentration difficulties, mood disorders, peripheral neuropathy characterized by numbness and tingling, muscular weakness and pain, joint discomfort, increased susceptibility to osteoporosis and associated fracture risks, gait disturbances, reproductive challenges including infertility, miscarriage, and premature or low birth weight deliveries, irregular menstrual patterns, onset or exacerbation of Type 2 diabetes, dyslipidemia or elevated cholesterol levels, heightened risk of kidney disease, anemia, and sleep apnea. In severe cases, untreated hypothyroidism may lead to myxedema coma, thyroid crisis, heart failure, respiratory failure, and septic shock

Diagnosis:

Diagnosis of hypothyroidism is based on clinical examination, assessment of Thyroid-stimulating hormone (TSH) levels, and evaluation of free thyroxine (T4) levels. Clinical manifestations may encompass an enlarged thyroid gland, weight gain, bradylalia and bradykinesia, xerosis, coarse and brittle hair, pallor or jaundice, impassive facial expressions, macroglossia, bradycardia, pericardial effusion, and prolonged ankle reflex relaxation time.

In primary hypothyroidism, diminished serum T4 levels and escalated thyroid-stimulating hormone (TSH) levels are observed. In secondary hypothyroidism, both free T4 and serum TSH levels are diminished (occasionally with normal TSH levels but reduced bioactivity). Subclinical hypothyroidism is characterized by elevated serum TSH levels and normal serum free thyroxine (T4) levels.

MCQ's

1. Which of the following is a common cause of hyperthyroidism
   a) Addison's disease b) Graves' disease
   c) Cushing's syndrome d) Hashimoto's thyroiditis

2. Which of the following is a clinical feature of hyperthyroidism
   a) Weight gain b) Cold intolerance
   c) Increased appetite d) Dry Skin

3. What severe complication can arise from untreated hyperthyroidism
   a) Thyroid storm b) Hypothyroidism
   c) Addisonian crisis d) Cushing syndrome

4. What thyroid profile test result is indicative of overt hyperthyroidism
   a) Elevated TSH, low free T4
   b) Low or suppressed TSH, elevated free T4 and total T3
   c) Normal TSH, elevated free T4
   d) Elevated TSH, elevated free T4

5. Which condition is characterized by a diffuse, non-nodular enlargement of the thyroid
   a) Toxic adenoma b) Graves' disease
   c) Toxic multinodular goitre d) Subacute thyroditis

6. What is the most prevalent cause of primary hypothyroidism in iodine-sufficient areas
   a) Iodine deficiency b) Hashimoto's thyroiditis
   c) Postpartum thyroiditis d) Subacute granulomatous thyroiditis

7. What is a common cause of secondary hypothyroidism
   a) Autoimmune thyroiditis b) Pituitary tumours
   c) Iodine deficiency d) Thyroid surgery

8. What hormone does the hypothalamus secrete to stimulate the pituitary gland in the thyroid hormone regulation process
   a) TSH (Thyroid-Stimulating Hormone) b) T4 (Thyroxine)
   c) T3 (Triiodothyronine) d) TRH (Thyrotropin-Releasing Hormone)

9. What is the typical thyroid profile test result for primary hypothyroidism
   a) Elevated TSH, low free T4 b) Low TSH, elevated free T4
   c) Normal TSH, elevated free T4 d) Elevated TSH, elevated free T4

10. Which of the following is a severe complication of untreated hypothyroidism
    a) Thyroid storm b) Myxedema coma
    c) Addisonian crisis d) Cushing syndrome

Answer Keys

1	b	2	c	3	a	4	b	5	b	6	b	7	b	8	d	9	a	10	b

Short Answer Questions

1. Explain Hyperthyroidism
2. Explain Hypothyroidism

◆

TOPIC 36

Sthaulya and Kārśya

Introduction

Sthaulya is one among the Śleṣmaja Nānātmaja Vyādhi and it is considered as Santarpaṇottha Vyadhi, whereas Kārśya is one among the Vātaja Nānātmaja Vyādhi and is considered as Apatarpaṇottha Vyādhi. Ācārya Caraka described and classified Sthaulya and Kārśya under the Aṣṭaunindītīya puruṣa in Sūtrasthāna. Ācārya Suśruta explained Sthaulya and Kārśya under the Rasanimittaja Vyādhi (dependent upon the Rasa dhātu) in Doṣadhātumalakṣayavṛddhivijñānīya adhyāya in Sūtrasthāna. Ācārya Vāgbhaṭa explained Sthaulya and Kārśya in Dvividhopakramaṇiya adhyāya in Sūtrasthāna and also he had mentioned that Kārśya is better than Sthaulya, because there is no treatment for Sthaulya.

Sthūla, Pīna, Pīva, Pīvaram, Atisthaulya, Medoroga, Medodoṣa and Jāṭhārya are used as synonyms of Sthaulya. Kṛśa, Atikārṣya and Atikṛśa are used as synonyms of Kārśya.

STHAULYA

Nirūkti and Paribhāṣa

The term 'Sthaulya' is derived from the root 'Sthū' with 'Ac' and 'Ṣyañ' pratyaya as suffixes, which means bulky or corpulent or stout.

- ◉ स्थूलस्य भावः स्थौल्यं । (SKD)

That which express the Sthūla bhāva (bulky or corpulent or stoutness) of the body is called Sthaulya.

- ◉ शरीरोपचयेत्यादि उपचयः स्थौल्यम् । (Ḍa.Su.Sū.10/5)

That which causes Upacaya (promotes or increases) of the body is called Sthaulya.

- ◉ स्थूलः उपचितावयवम् । (SKD)

That which causes Upacitāvayava (promotes or increases the part) of the body is called Sthaulya or Sthūla.

- ◉ मेदोमांसातिवृद्धत्वाच्चलस्फिगुदरस्तनः। अयथोपचयोत्साहो नरोऽतिस्थूल उच्यते॥ (Ca.Sū.21/9)

That which causes Calasphigudarastana (pendulous appearance of buttocks, abdomen and breasts) due to excessive increase of Medas (fat) and Māṃsa (muscle) is called as Atisthūla or Sthaulya in which there will be reduced Upacayotsāha (nourishment and enthusiasm).

- ◉ मेदःसङ्कीर्तनात् मेदोदुष्टेरभिधानं, मेदोदुष्ट्या च स्थौल्यम् । (Ma.M.N.34/1-9)

Due to Medodhātu duṣṭi, there is excessive accumulation of Medodhātu in the body resulting in the disease Sthaulya.

- ◉ मेदोवृद्धिजनितो दोषो मेदोदोषः । (Āḍ.Śā.P.7/65)

That which causes Medovṛddhi (increase of fat) in the body is called Medodoṣa.

Nidāna and Samprāpti

- ◉ तत्र श्लेष्मलाहारसेविनोऽध्यशनशीलस्याव्यायामिनो दिवास्वप्नरतस्य चाम एवान्नरसो मधुरतरश्च शरीरमनुक्रामन्नतिस्नेहान्मेदो जनयति, तदतिस्थौल्यमापादयति । (Su.Sū.15/32)

In person indulging in Śleṣmāhāra (diet which promotes the Kapha), Adhyaśana (those who intake food before the digestion of previous food), Avyāyāma (lack of physical exercise), Divāsvapna (sleeping during day-time), the Āhāra rasa being in Āmāvastha (undigested stage) and remains Madhura (sweet) circulating in the body, due to Atisnehān (excessive unctuousness), there will be formation of excessive Medas resulting in Sthaulya.

- ◉ तदतिस्थौल्यमतिसम्पूरणाद्गुरुमधुरशीतस्निग्धोपयोगादव्यायामादव्यवायाद्दिवास्वप्नाद्धर्षनित्यत्वादचिन्तनाद्बीजस्वभावाच्चोपजायते। (Ca.Sū.21/4)

Atisthaulya (excessive obesity) is caused due to Atisampūraṇād (Atibhojanāt – excessive intake of food), Gurumadhuraśītasnigdhopayogat (intake of a heavy, sweet, cold and fatty diet), Avyāyāma (lack of physical exercise), Avyavāya (absence of sexual act), Divāsvapnāt (sleeping during day-time), Harṣanityātvāt (continuos happiness), Acintanāt (absence of mental worries), and Bījasvabhāvat (genetic defects) leads to excessive accumulation of Medas and produces the disease Sthaulya.

- ◉ मेदसाऽऽवृतमार्गत्वाद्वायुः कोष्ठे विशेषतः। चरन् सन्धुक्षयत्यग्निमाहारं शोषयत्यपि॥
  तस्मात् स शीघ्रं जरयत्याहारं चातिकाङ्क्षति। विकारांश्चाश्नुते घोरान् कांश्चित्कालव्यतिक्रमात्॥
  एतावुपद्रवकरौ विशेषादग्निमारुतौ। एतौ हि दहतः स्थूलं वनदावो वनं यथा॥
  मेदस्यतीव संवृद्धे सहसैवानिलादयः। विकारान् दारुणान् कृत्वा नाशयन्त्याशु जीवितम्॥ (Ca.Sū.21/5-8)

The accumulated Medas obstructs the passage of Vāta resulting in aggravation of Vāta especially in the Koṣṭha afflicting the Agni (digestive fire) and does Āhāra śoṣa (absorption of food) resulting in Śīgraṃ jarayatyāhāraṃ (food gets digested quickly) and Atikāṅkṣati (excessive desire for food). However, if food intake is delayed, then the person is afflicted with dreadful diseases. Specifically Agni and Vāta are the two factors responsible for producing the Upadravā (complications) in case of Sthaulya and these factors burns the Sthūla person like forest fire burning the forest. Due to excessive increase of Medas and sudden vitiation of Vāta etc., dreadful disease which may lead to immediate death occurs.

Saṃprāpti Ghaṭaka of Sthaulya

Doṣa	Kapha Pradhāna Tridoṣas
Dūṣya	Māṃsa and Medas
Agni	Jaṭharāgni Medo Dhātvāgni māndhya
Āma	Agni māndhya janya Āma
Srotas	Rasa, Māṃsa and Medovaha
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Sarva śārīra
Adhiṣṭhāna	Medodharā kalā, Vapavahana
Udbhava sthāna	Āmāśaya
Vyakta sthāna	Sarvāṅga especially Sphik, Udara and Stana
Rogamārga	Bāhya

Sthaulya Bheda

- तत्र संशोधनैः स्थौल्यबलपित्तकफाधिकान्। आमदोषज्वरच्छर्दिरतीसारहृदामयैः॥
  विबन्धगौरवोद्गारहृल्लासादिभिरातुरान्। मध्यस्थौल्यादिकान् प्रायः पूर्वं पाचनदीपनैः॥
  एभिरेवामयैरार्तान् हीनस्थौल्यबलादिकान्। क्षुत्तृष्णानिग्रहैर्दोषैस्त्वार्तान् मध्यबलैर्दृढान्॥
  समीरणातपायासैः किमुताल्पबलैर्नरान्। (A.H.Sū.14/12-15)

Ācārya Vāgbhaṭa classified Sthaulya into three types namely Hīna Sthaulya, *Madhya Sthaulya* and Atisthaulya in the Dvividhopakramaṇiya adhyāya in Sūtrasthāna while *describing the Laṅghana therapy.*

- In case of **Atisthaulya**, having predominance of Pitta and Kapha, and those afflicted with Āmadoṣa, Jvara, Chardi, Atisāra, Hṛdāmaya, Vibandha, Gaurava, Udgāra and Hṛllāsa etc., Saṃśodhana (purificatory therapies) should be administered.
- In case of **Madhyasthaulya**, Pācana (digestant) and Dīpana (appetizers) should be advocated initially.
- In case of **Hīnasthaulya**, Laṅghana (fasting therapy) should be advocated especially in the method of Kṣuttṛṣṇānigraha (suppressing the hunger and thirst), if the person is Dṛḍān (strong) and has Doṣairārtān madhyabala (moderate vitiation of Vātādi doṣas), then Samīraṇātapāyāsa (exposure to breeze, sunlight and exercise) should be advocated.

Sāmānya Lakṣaṇās of Sthaulya

- मेदोमांसातिवृद्धत्वाच्चलस्फिगुदरस्तनः। अयथोपचयोत्साहो नरोऽतिस्थूल उच्यते॥ (Ca.Sū.21/9)

Calasphigudarastana (pendulous appearance of buttocks, abdomen and breasts) due to excessive increase of Medas (fat) and Māṃsa (muscle) and reduced Upacayotsāha (nourishment and enthusiasm) are the features of Atisthūla or Sthaulya.

- अतिस्थूलस्य तावदायुषो ह्रासो जवोपरोधः कृच्छ्रव्यवायता दौर्बल्यं दौर्गन्ध्यं स्वेदाबाधः क्षुदतिमात्रं पिपासातियोगश्चेति भवन्त्यष्टौ दोषाः। (Ca.Sū.21/4)

Āyuṣa hrāso (reduced lifespan), Javoparodhaḥ (restricted movement), Kṛcchravyavāya (difficulty in sexual activities), Daurbalya (weakness), Daurgandhya (bad odour), Svedābādha (excessive perspiration), Kṣudatimātraṃ (excessive hunger) and Pipāsātiyogāt (excessive thirst) are the eight doṣās of Atisthūla or Sthaulya.

- तमतिस्थूलं क्षुद्रश्वासपिपासाक्षुत्स्वप्नस्वेदगात्रदौर्गन्ध्यक्रथनगात्रसादगद्गदत्वानि क्षिप्रमेवाविशन्ति। (Su.Sū.15/32)

Kṣudra śvāsa (dyspnoea on exertion), Pipāsa (thirst), Kṣut (excessive hunger), Svapna (excessive sleepiness), Sveda (excessive perspiration), Gātradaurgandhya (bad odour from the body), Krathana (snoring), Gātrasāda (weakness of the body) and Gadgada (Avyaktavacanatvam – indistinctness of speech) are the features developing immediately in case of Atisthūla or Sthaulya.

- सौकुमार्यान्मेदसः सर्वक्रियास्वसमर्थः, कफमेदोनिरुद्धमार्गत्वाच्चाल्पव्यवायो भवति, आवृतमार्गत्वादेव शेषा धातवो नाप्यायन्तेऽत्यर्थमतोऽल्पप्राणो भवति, प्रमेहपिडकाज्वरभगन्दर विद्रधिवातविकाराणामन्यतमं प्राप्य पञ्चत्वमुपयाति, सर्व एव चास्य रोगा बलवन्तो भवन्त्यावृतमार्गत्वात् स्रोतसाम्; अतस्तस्योत्पत्तिहेतुं परिहरेत्। (Su.Sū.15/32)

Due to Saukumāryānta medas (softness of fat), the Sthūla person becomes Sarvakriyāsvasamarthaḥ (incapable of doing all physical activities); due to Kaphamedoniruddhamārga (the obstruction of channels by Kapha and Medas), they become Alpavyavāyo bhavati (less active in sexual act); Āvṛtamārgatvādeva śeṣā dhātavonāpyāyante atyarthamatoalpaprāṇo bhavati (due to obstruction of passages, the nourishment of dhātus is decreased, thus they become weak) and they get afflicted with the diseases such as Prameha Piḍakā, Jvara, Bhagandara, Vidradhi and Vātavikārās resulting in Pañcatvamupayāti (approaching towards death). All diseases occurring in them becomes Balavanta (strong) due to Āvṛtamārgatvāt srotasām (obstruction in the passage of Srotas), hence one should avoid all the Hetu (causative factors).

Upadravā of Sthaulya

- ⊙ प्रमेहपिडकाज्वरभगन्दर विद्रधिवातविकाराणामन्यतमं प्राप्य पञ्चत्वमुपयाति, । (Su.Sū.15/32)
- ⊙ विकारान् दारुणानिति प्रमेहपिडकाज्वरभगन्दरविद्रधिवातरोगाणामन्यतमान्। (Ma.M.N.34/1-2)
- ⊙ अतिस्थौल्यापचीमेहज्वरोदरभगन्दरान्। काससन्न्यासकृच्छ्रामकुष्ठादीनतिदारुणान्॥ (A.H.Sū.14/20)
- ⊙ स्थूले स्युर्दुस्तराः कुष्ठा विसर्पाः सभगन्दराः ज्वरातीसारमेहार्श श्लीपदापचिकाम्लाः । मेदसः स्वेददौर्गन्ध्याज्जायन्ते जन्तवोऽणवः ॥ (B.P.M.39/10)

Sthaulya Upadravā	Suśruta	Vāgbhaṭa	Mādhavakara	Bhāvaprākāśa
Prameha Piḍakā	+	−	+	−
Jvara	+	+	+	+
Bhagandara	+	+	+	+
Vidradhi	+	−	+	−
Vātavikārās	+	−	+	−
Apacī	−	+	−	−
Meha	−	+	−	+
Udara	−	+	−	−
Kāsa	−	+	−	−
Sanyāsa	−	+	−	−
Mūtrakṛcchra	−	+	−	−
Āma	−	+	−	−
Kuṣṭha	−	+	−	+
Visarpā	−	−	−	+
Atisāra	−	−	−	+
Arśas	−	−	−	+
Ślīpada	−	−	−	+
Kāmalā	−	−	−	+
Jantavoaṇavaḥ	−	−	−	+

KĀRŚYA

Nirūkti and Paribhāṣa

The word 'Kārśya' is derived from the word 'Kṛśa' meaning 'Tanūkaraṇe' which means making thin or emaciated. Hence, the word 'Kārśya' means emaciation of the body.

- ⊙ कृशस्य भावः कार्श्य । (SKD)

That which expresses the Kṛśa bhāva (thinness or leanness or emaciation) of the body is called Kārśya.

- ⊙ कार्श्य कृशकरणाम् ॥ (SKD)

That which produces Kṛṣa (thinness or leanness or emaciation) of the body is called Kārśya.

- ⊙ अपचय कार्श्यः । (Ḍa.Su.Sū.10/5)

That which causes Apacaya (diminution or decreases) of the body is called Kārśya.

- ⊙ कार्श्य मांसक्षयः। (Ḍa.Su.Sū.15/13)

That which causes Māṃsa kṣaya (diminution of muscles) is called Kārśya.

- ⊙ कार्श्यमिति कृशता शरीरस्य वातप्रकृतिजत्वात् ॥ (Āḍ.Śā.P.7/112)

That which causes Kṛśata śarīrasya (emaciation of the body) is called Kārśya and it is due to Vāta.

- ⊙ शुष्कस्फिगुदरग्रीवो धमनीजालसन्ततः। त्वगस्थिशेषोऽतिकृशः स्थूलपर्वा नरो मतः॥ (Ca.Sū.21/15)

That which causes Śuṣkasphigudaragrīva (emaciated buttocks, abdomen and neck), Dhamanījālasantataḥ (prominent network of vessels all over the body) and Tvagasthiśeṣo sthūla parvā (skinny and bony body with thickened joints) is called as Atikṛśa or Kārśya.

Nidāna and Saṃprāpti

- ⊙ सेवा रूक्षान्नपानानां लङ्घनं प्रमिताशनम्। क्रियातियोगः शोकश्च वेगनिद्राविनिग्रहः॥
  रूक्षस्योद्वर्तनं स्नानस्याभ्यासः प्रकृतिर्जरा। विकारानुशयः क्रोधः कुर्वन्त्यतिकृशं नरम्॥ (Ca.Sū.21/11-12)

Indulging in Rūkṣānnapāna (non-unctuous diet and drinks), Laṅghana (fasting), Pramitāśana (intake of little food), Kriyātiyoga (excess application of purificatory therapies), Śoka (grief), Veganidrāvinigrahaḥ (suppressing the urges including sleep), Rūkṣasyodvaratana (non-unctuous anointments), Snānasyābhyāsa (bath), Prakṛti (genetic factor), Jarā (old age), Vikārānuśaya (prolonged illness) and Krodha (anger) are the causative factors that makes the person Atikṛśa.

- तत्र पुनर्वातलाहारसेविनोऽतिव्यायामव्यवायाध्ययनभयशोकध्यानरात्रिजागरणपिपासाक्षु त्कषायाल्पाशनप्रभृतिभिरुपशोषितो रसधातुः शरीरमननुक्रामन्नल्पत्वान्न प्रीणाति, तस्मादतिकार्श्यं भवति; (Su.Sū.15/33)

Indulging in Vātalāhāra (diet which promotes the Vāta), Ativyāyāma (excessive physical exercise), Vyavāya (excessive sexual intercourse), Adhyayana (excessive study), Bhaya (fear), Śoka (grief), Rātrijāgaraṇa (vigil), Pipāsā (thirst), Kṣut (hunger), Kaṣāya (astringent taste) and Alpāśana (less intake of food) leads to Rasa dhātu śoṣaṇa (reduces the quantity of Rasa dhātu) in sequence reducing the nourishment to the body, thus making the person Atikārśya.

Samprāpti Ghaṭaka of Kārśya

Doṣa	Vāta Pradhāna Tridoṣas
Dūṣya	Rasa
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rasa, Māṃsa and Medovaha
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Sarva Rasāyanīs
Adhiṣṭhāna	Sarva Śarīra
Udbhava sthāna	Āmapakvāśaya
Vyakta sthāna	Sarvāṅga especially Sphik, Udara and Grīva
Rogamārga	Bāhya

Sāmānya Lakṣaṇās of Kārśya

- शुष्कस्फिगुदरग्रीवो धमनीजालसन्ततः। त्वगस्थिशेषोऽतिकृशः स्थूलपर्वा नरो मतः॥ (Ca.Sū.21/15)

Śuṣkasphigudaragrīva (emaciated buttocks, abdomen and neck), Dhamanījālasantataḥ (prominent network of vessels all over the body) and Tvagasthiśeṣo sthūla parvā (skinny and bony body with thickened joints) are the clinical features of Atikṛśa or Kārśya.

- सोऽतिकृशः क्षुत्पिपासाशीतोष्णवातवर्षभारादानेष्वसहिष्णुर्वातरोगप्रायोऽल्पप्राणश्च क्रियासु भवति, श्वासकासशोषप्लीहोदराग्निसादगुल्मरक्तपित्तानामन्यतममासाद्य मरणमुपयाति, सर्व एव चास्य रोगा बलवन्तो भवन्त्यल्पप्राणत्वात्; अतस्तस्योत्पत्तिहेतुं परिहरेत् । (Su.Sū.15/33)

Kṣutpipāsaśītoṣṇavātavarṣa bhārādāneṣvasahiṣṇuta (intolerance to hunger, thirst, cold, hot, breeze, rain and weight-carrying) and Vātarogaprāyo (afflicted mostly with Vātaja disorders) and Alpaprāṇaśca kriyāsu bhavati (low vitality in all activities) are the features of Kārśya and in sequence, they may suffer from Śvāsa, Kāsa, Kṣaya, Plīha, Udara, Agnisāda, Gulma, Raktapitta and finally it leads to Marana (death). All diseases occurring in them becomes Balavanta (strong) due to Alpaprāṇatvāt (low vitality), hence one should avoid all the Hetu (causative factors).

- व्यायाममतिसौहित्यं क्षुत्पिपासामयौषधम् । कृशो न सहते तद्वदतिशीतोष्णमैथुनम्॥
  प्लीहा कासः क्षयः श्वासो गुल्मोऽर्शांस्युदराणि च। कृशं प्रायोऽभिधावन्ति रोगाश्च ग्रहणीगताः॥ (Ca.Sū.21/13-14)

The Kṛśa person is Na sahate (intolerant) to Vyāyāma (exercise), Atisauhityaṃ (over nutrition), Kṣut (hunger), Pipāsa (thirst), Āmaya (diseases) and Auṣadha (drugs) and also for Atiśītoṣṇamaithuna (too much cold, heat and sexual intercourse). Such a person is often suffers from Plīha, Kāsa, Kṣaya, Śvāsa, Gulma, Arśas, Udara and Grahaṇī.

- कश्चिदन्यः कृशोऽतीव बलवान्दृश्यते तदा। आधानसमये यस्य शुक्रभागोऽधिको भवेत्॥
  मेदोभागस्तु हीनः स्यात्स कृशोऽपि महाबलः। मेदसस्त्वधिका यस्य शुक्रभागोऽल्पको भवेत्॥
  स स्निग्धोऽपि सुपुष्टोऽपि बलहीनो विलोक्यते॥ (B.P.M.40/5-6)

Ācārya Bhāvamiśra described that during the time of formation of foetus, if the Śukra dhātu is Adhika (high) and the Medodhātu is Hīna (low), then the born child appears Kṛśa (emaciated) but has Mahābala (great strength); whereas if the Śukra dhātu is Alpa (low) and Meda dhātu is Adhika (high), then the born child appears Snigdha (unctuous) and Supuṣṭi (well nourished) but has Balahīna (weak strength)

Sādhyāsādhyata of Sthaulya and Kārśya

- कार्श्यमेव वरं स्थौल्यात् न हि स्थूलस्य भेषजम्॥ बृंहणं लङ्घनं वाऽलमतिमेदोग्निवातजित्। (A.H.Sū.14/31)
- स्थौल्यात् कार्श्यस्य सुखसाध्यत्वमाह । (In.A.H.Sū.14/31)

Comparatively Kārśya is Sukhasādhya (easily curable) than Sthaulya, because there is no treatment for Sthaulya; neither Bṛṃhaṇa nor Laṅghana are enough to control Atimeda (excessive fat), Agni (digestive fire) and Vāta.

- स्वभावादतिकार्श्यो यः स्वभावादल्पपावकः । स्वभावादबलो यश्च-तस्य नास्ति चिकित्सितम् ॥ (B.P.M.40/11)

There is no treatment to Kārśya if it occurs due to Svābhāva (nature or birth), Svabhāvādalpapāvakaḥ (whose digestive fire is weak) and Svabhāvādabalo (whose strength is low).

Differential diagnosis of Kārśya

Kārśya is one among the Aṣṭanindita, and it comes under Apatarpaṇajanya Vyādhi as well as it is a Rasapradoṣaja Vikāra. It is characterised by Śuṣkasphigudaragrīva (emaciated buttocks, abdomen and neck), Dhamanījālasantataḥ (prominent network of vessels all over the body) and Tvagasthiśeṣo sthūla parvā (skinny and bony body with thickened joints). The differential diagnosis of Kārśya necessitates the distinction from other conditions presenting similar symptoms.

- **Māṃsa Śoṣa** leads to Sphiggrīvodara śuṣkatā (dryness or wasting of muscles in the buttocks, neck and abdomen) when it manifests as Māṃsa kṣaya.
- **Kṣayaja Yakṣma** leads to Atikarśana (excessive emaciation) caused by Īrṣyā (envy or greed), Utkaṇṭhā (anxiety), Bhaya (fear), Trāsa (frightening), Krodha (anger), Śoka (grief), Ativyavāya (excessive sexual intercourse) and Anaśana (fasting) leading to the depletion of Śukra and Ojas (Rasa).
- **Bālāśoṣa** results in the Kumāraḥ śuṣyati (emaciation of child) along with the features such as Arocaka (anorexia), Pratiśyāya (coryza), Jvara (fever), Kāsa (cough) and Snigdhaśuklamukhekṣaṇaḥ (unctuous and paleness of face and eyes)

Kārśya may act as a causative factor for other conditions like Udara, Hikkā, Atisāra, Gulma, Śoṣa, and Klaibya. Furthermore, it can be observed as a symptom in various disorders such as Sannipātaja Jvara, Unmāda, Udara, Grahaṇī, Kāsa, Kṛmī, Arśas, and others. It may serve as a premonitory symptom of conditions like Arśas and Udara, or as a complication of chronic diseases as Vyādhi karśitā

In Contemporary science,

Sthaulya and Kārśya can be predominantly characterized by conditions like nutrition disorder in contemporary science. Exactly Sthaulya can be correlated with over nutrition diseases like obesity, hyperlipidaemia and hypervitaminosis. Whereas Kārśya can be correlated with under nutrition or malnutrition diseases like emaciation, marasmus in children, starvation, bulimia in adults, protein energy malnutrition (PEM), etc

Short Answer Questions

1. Define Sthaulya and explain the Saṃprāpti of Sthaulya
2. Describe Sthaulya bheda and its lakṣaṇās
3. Define Kārśya and explain the Saṃprāpti of Kārśya
4. Describe Sāmānya Lakṣaṇās of Kārśya

Long Answer Questions

1. Explain Sthaulya in detail
2. Explain Kārśya in detail

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TOPIC 37

Obesity

Introduction

Obesity is a chronic and complex disease characterized by the excessive accumulation of body fat, which has adverse implications for health, as per the World Health Organization (WHO). The classification of obesity is indicated by a Body Mass Index (BMI) of 30 or higher, while additional metrics such as waist circumference and waist-to-hip ratio are utilized to measure health risks associated with obesity. Globally, the prevalence of obesity has surged, with an estimated 16% of adults affected in 2022. By 2023, approximately 24% of women and 22.9% of men in India were classified as obese.

The etiology of obesity includes a multitude of factors, including genetic predisposition, behavioral influences (diet, physical activity), metabolic factors (hormonal imbalances, insulin resistance), environmental factors (sedentary lifestyle, poor sleep, chronic stress), and socioeconomic factors (access to unhealthy foods, limited physical activity opportunities). Contributing factors also encompass reduced physical activity, insomnia, endocrine disorders, medications, decreased energy metabolism, and high consumption of carbohydrates and sugary foods. Genetic syndromes associated with obesity include Prader-Willi, MC4R, fragile X, Bardet-Biedl, Wilson-Turner, congenital leptin deficiency, and Alstrom.

Pathophysiology:

The pathophysiology of obesity involves a complex interplay of genetic, environmental, and physiological factors. The hypothalamus, a key regulatory center, governs hunger and energy expenditure through the modulation of leptin and ghrelin signals. Adipose tissue, functioning as an endocrine organ, releases adipokines and inflammatory cytokines, culminating in chronic inflammation, insulin resistance, and metabolic disturbances.

Genetic predisposition and epigenetic changes significantly affect appetite regulation, fat storage and energy expenditure. Hormonal imbalances, particularly insulin resistance and leptin resistance, further exacerbate the condition. An imbalance in gut microbiota influences energy extraction from food and fat storage. Behavioural and environmental factors, including high-calorie diets, sedentary lifestyles, poor sleep, and chronic stress, significantly contribute to obesity.

Clinical features

Obesity is characterized by excess body fat, particularly concentrated around the abdomen, hips, and thighs, with increased waist circumference (men: ≥40 inches, women: ≥35 inches) and weight gain despite weight loss attempts. Physical symptoms include fatigue, decreased mobility and flexibility, joint pain and stiffness, sleep disturbances, decreased libido, and menstrual irregularities.

- Systemic symptoms include hypertension, dyslipidemia, increased risk of cardiovascular disease, and shortness of breath.
- Metabolic symptoms include insulin resistance and type 2 diabetes, hyperglycemia, dyslipidemia, and fatty liver disease.
- Dermatological symptoms comprise skin folds and creases, acne, hirsutism, stretch marks, and poor wound healing.
- Psychological symptoms include depression, anxiety, body dysmorphic disorder, and eating disorders.

Additional symptoms include gastroesophageal reflux disease, urinary incontinence, erectile dysfunction, polycystic ovary syndrome, and increased risk of certain cancers. Complications associated with obesity encompass cardiovascular disease, type 2 diabetes, stroke, sleep apnea, osteoarthritis, and mental health disorders.

Diagnosis

Diagnosis is based on medical history, physical examination, BMI calculation, waist circumference measurement, and body composition analysis. A comprehensive medical history is essential in assessing obesity, involving inquiries pertaining to family history, dietary habits, physical activity, medical conditions (such as diabetes, hypertension, and sleep apnea), medications (such as steroids, antidepressants), previous weight loss attempts, and psychological factors (such as stress, depression). This helps identify potential causes and consequences of obesity.

A thorough physical examination assesses body mass index (BMI), waist circumference, blood pressure, heart rate, respiratory rate, body fat distribution, and signs of insulin resistance. BMI calculation, using the formula weight (kg) / height $(m)^2$, categorizes individuals as

- **Underweight:** less than 18.5 kg/m2
- **Normal range:** 18.5 kg/m2 to 24.9 kg/m2

- **Overweight:** 25 kg/m2 to 29.9 kg/m2
- **Obese, Class I:** 30 kg/m2 to 34.9 kg/m2
- **Obese, Class II:** 35 kg/m2 to 39.9 kg/m2
- **Obese, Class III:** more than 40 kg/m2

Waist circumference measurement, taken at the midpoint between the iliac crest and lower rib, assesses central obesity. Men with a waist circumference ≥40 inches (102 cm) and women ≥35 inches (88 cm) are at increased risk.

Body composition analysis like skinfold thickness, bioelectric impedance analysis, and underwater (hydrostatic) weighing can be helpful in measuring the percentage of body fat. Additionally, imaging studies such as Computed Tomography (CT), Magnetic Resonance Imaging (MRI) and dual-energy x-ray absorptiometry (DEXA) studies helps to measure body fat percentage, lean body mass, visceral fat area, muscle mass and bone density.

Differential diagnosis of Obesity

A differential diagnosis for obesity includes various conditions, such as endocrine disorders (Hypothyroidism, Cushing's syndrome, Polycystic ovary syndrome (PCOS), Growth hormone deficiency, and Insulinoma); genetic disorders (Prader-Willi syndrome, Bardet-Biedl syndrome, and Leptin receptor deficiency); neurological disorders (Hypothalamic lesions, Brainstem lesions, and Kleine-Levin syndrome); medication-induced weight gain (Psychiatric drugs, corticosteroids, insulin, and specific β-adrenergic receptor blockers); other medical conditions (Sleep apnea, Diabetes, fatty liver, gastroesophageal reflux disease (GERD), hirsutism, and polygenic hypercholesterolemia); and nutritional and lifestyle factors (Poor diet, Sedentary lifestyle, Alcoholism, and Stress).

MCQ's

1. Which of the following is a common genetic syndrome associated with obesity

a) Down syndrome	b) Prader-Willi syndrome
c) Turner syndrome	d) Klinefelter syndrome

2. What hormone, produced by adipose tissue, plays a crucial role in regulating hunger and energy expenditure

a) Insulin	b) Ghrelin
c) Leptin	d) Cortisol

3. What is the BMI threshold for defining obesity according to the World Health Organization (WHO)
   a) 25 b) 30
   c) 35 d) 40
4. Which of the following is a metabolic complication commonly associated with obesity
   a) Osteoporosis b) Type 2 diabetes
   c) Hyperthyroidism d) Anemia
5. What is the primary role of the hypothalamus in the pathophysiology of obesity
   a) Regulating blood pressure
   b) Controlling hunger and energy expenditure
   c) Managing sleep cycles
   d) Producing digestive enzymes
6. Which of the following is a psychological symptom commonly associated with obesity
   a) Increased libido b) Anxiety
   c) Improved sleep d) Enhanced mobility
7. What waist circumference measurement is considered a risk factor for obesity-related health issues in men
   a) ≥30 inches b) ≥35 inches
   c) ≥40 inches d) ≥45 inches
8. Which of the following is NOT a common complication of obesity
   a) Cardiovascular disease b) Type 2 diabetes
   c) Osteoarthritis d) Hyperthyroidism

What is the role of the gut microbiota in the pathophysiology of obesity
   a) Regulating blood pressure b) Influencing energy extraction from food
   c) Controlling body temperature d) Enhancing muscle growth

Which of the following medications is commonly associated with weight gain
   a) Metformin b) Corticosteroids
   c) ACE inhibitors d) Beta-blockers

11. Which of the following is a dermatological symptom associated with obesity
    a) Alopecia b) Acne
    c) Psoriasis d) Vitiligo
12. Which of the following is a common complication of obesity in the respiratory system
    a) Asthma b) Chronic bronchitis
    c) Sleep apnea d) Tuberculosis
13. Which of the following is a key feature of metabolic syndrome often associated with obesity
    a) Hyperthyroidism b) Hyperglycemia
    c) Hypotension d) Hypoglycemia
14. What is the formula for calculating Body Mass Index (BMI)
    a) Weight (kg) / height (m) b) Weight (kg) / height $(m)^2$
    c) Height (m) / weight (kg) d) Height $(m)^2$ / weight (kg)
15. What waist circumference measurement is considered a risk factor for obesity-related health issues in women?
    a) ≥30 inches (76 cm) b) ≥35 inches (89 cm)
    c) ≥40 inches (102 cm) d) ≥45 inches (114 cm)

Answer Keys

1	b	2	c	3	b	4	b	5	b	6	b	7	c	8	d
9	b	10	b	11	b	12	c	13	b	14	b	15	b		

Short Answer Questions

1. Explain Obesity in detail
2. Describe Obesity with its diagnostic features

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TOPIC 38

Prameha

Introduction

Prameha is defined as Āvilaprabhūtamūtratā (turbidity and excessive urination) due to involvement of Kaphapradhāna tridoṣas and Dāśadūṣyās (Rasa, Rakta, Māṃsa, Meda, Majjā, Śukra, Vasā, Ojas, Lasīkā and Kleda). Hence, a group of urinary disorders including diabetes mellitus which has Avilaprabhūtamūtratā (turbidity and excessive urination) comes under the disease Prameha. Ācārya Caraka explained Prameha after Gulma because, the disease Prameha manifested due to intake of Havi (substances which are thrown in fire of Yajña) after Gulma during the destruction of Dakṣās yajña. And also he considered that Prameha is a Kulaja vikāra (hereditary disease), Anuṣaṅgī (i.e. a recurring disease (afflicts the patient continuously), and one among the Aṣṭa mahāgada (eight dreadful diseases).

Ācārya Suśruta described Prameha after Kuṣṭha because of their similarity in Apatyasaṃkramyatva (transferring to offspring), Tridoṣārabdhatva (commencing with involvement of Tridoṣas), Mahāvyādhitva (dreadful or major diseases), Kālāntareṇāsādhyatva (becomes incurable after some time) and Sādharmyāccikitsaikatva (homogenous in the treatment).

Nirūkti and Paribhāṣa

The word Prameha is a combination of two words i.e. 'Pra (upasarga) and Meha (dhātu)'. The word 'Meha' is derived from the root 'Miha Secane' which is employed in the sense of Siñcana (to moisten), Kṣarana (to flow) and Prasrāva (to flow excessively) in terms of urine. By adding the prefix 'Pra', it illustrates excessive in both frequency and quantity.

- प्रकर्षेण मेहति क्षरति वीर्य्यादिरनेनेति । (SKD)

That which causes excessive excretion of the urine associated with flow out of energy etc. is known as Prameha.

- प्रकर्षेण मेदोमांसक्लेदादिमेहतीति प्रमेहः ॥ (Āḍ.Śā.P.7/59-62)

That which causes excessive excretion of the Medas (fat), Māṃsa (muscle), Kleda (moistureness of the body) etc. along with urine is known as Prameha.

Bheda of Prameha

- प्रमेहा विंशतिस्तत्र श्लेष्मतो दश, पित्ततः। षट्, चत्वारोऽनिलात्..... । (A.H.Ni.10/1)

Prameha is classified into twenty types based on the predominance of doṣas i.e. Kaphaja Pramehās are ten in number, Pittaja Pramehās are six in number and Vātaja Prameha Pramehās are four in number.

- ते तु खल्विमे दश प्रमेहा नामविशेषेण भवन्ति; तद्यथा- उदकमेहश्च, इक्षुवालिकारसमेहश्च, सान्द्रमेहश्च, सान्द्रप्रसादमेहश्च, शुक्लमेहश्च, शुक्रमेहश्च, शीतमेहश्च, सिकतामेहश्च, शनैर्मेहश्च, आलालमेहश्चेति॥ (Ca.Ni.4/10)
- तेषामपि तु खलु पित्तगुणविशेषेणैव नामविशेषा भवन्ति; तद्यथा- क्षारमेहश्च, कालमेहश्च, नीलमेहश्च, लोहितमेहश्च, माञ्जिष्ठमेहश्च, हारिद्रमेहश्चेति॥ (Ca.Ni.4/25)
- तेषामपि पूर्ववद्गुणविशेषेण नामविशेषा भवन्ति; तद्यथा- वसामेहश्च, मज्जमेहश्च, हस्तिमेहश्च, मधुमेहश्चेति॥ (Ca.Ni.4/39)
- तत्र, कफादुदकेक्षुवालिकासुरासिकताशनैर्लवणपिष्टसान्द्रशुक्रफेनमेहा दश साध्याः, दोषदूष्याणां समक्रियत्वात्; पित्तान् नीलहरिद्राम्लक्षारमञ्जिष्ठाशोणितमेहाः षड् याप्याः, दोषदूष्याणां विषमक्तियत्वात्; वातात् सर्पिर्वसाक्षौद्रहस्तिमेहाश्चत् वारोऽसाध्यतमाः, महात्ययिकत्वात् ॥ (Su.Ni.6/8)

Kaphaja Prameha	Caraka	Suśruta	Vāgbhaṭa
Udakameha	+	+	+
Ikṣuvālikameha	+	+	+
Sāndrameha	+	+	+
Sāndraprasādameha	+	–	–
Śuklameha	+	–	–
Śukrameha	+	+	+
Śītameha	+	–	+
Sikatāmeha	+	+	+
Śanairmeha	+	+	+
Ālālameha	+	–	+
Surāmeha	–	+	+
Lavaṇameha	–	+	–
Piṣṭameha	–	+	+
Phena meha	–	+	–

Pittaja Prameha	Caraka	Suśruta	Vāgbhaṭa
Nīlameha	+	+	+

Hāridrameha	+	+	+
Mañjiṣṭhāmeha	+	+	+
Lohitameha / Śoṇitameha / Raktameha	+	+	+
Kṣārameha	+	+	+
Kālameha	+	–	+
Amlameha	–	+	–
Vātaja Prameha	**Caraka**	**Suśruta**	**Vāgbhaṭa**
Vasāmeha	+	+	+
Majjāmeha	+	–	+
Hastimeha	+	+	+
Madhumeha/ Kṣaudrameha	+	+	+
Sarpimeha	–	+	–

◉ तत्र वातपित्तमेदोभिरन्वितः श्लेष्मा श्लेष्मप्रमेहाञ्जनयति, वातकफशोणितमेदोभिरन्वितं पित्तं पित्तप्रमेहान्, कफपित्तवसामज्जमेदोभिरन्वितो वायुर्वातप्रमेहान् ॥ (Su.Ni.6/9)

◈ **Kaphaja Prameha** occurs due to combination of Kapha with the Pitta, Vāta and Medas;

◈ **Pittaja Prameha** occurs due to combination of Pitta with Vāta, Kapha, Śoṇita and Medas; and

◈ **Vātaja Prameha** occurs due to combination of Vāta with Kapha, Pitta, Vasā, Majja and Medas.

◉ स्थूलः प्रमेही बलवानिहैकः कृशस्तथैकः परिदुर्बलश्च। (Ca.Ci.6/15)

Ācārya Caraka while describing the treatment, he classified Prameha into two types based on the physical strength, i.e. Sthūla and Kṛśa. Sthūla pramehi is Balavān (strong) whereas Kṛśa pramehi is Paridurbalaḥ (weak).

◉ साध्ययाप्यपरित्याज्या मेहास्तेनैव तद्भवाः। समासमक्रियतया महात्ययतयाऽपि च॥ (A.H.Ni.10/6)

Ācārya Vāgbhaṭa classified Prameha into three types based on the prognosis i.e. Sādhya (curable), Yāpya (difficult to treat or controllable) and Parityājyā (incurable or rejectable) because of Samakriyatā (identical therapy), Asamakriyatā (non-identical therapy) and Mahātyayatā (severity involved).

◉ दृष्ट्वा प्रमेहं मधुरं सपिच्छं मधूपमं स्याद्द्विविधो विचारः। सम्पूरणाद्वा कफसम्भवः स्यात् क्षीणेषु दोषेष्वनिलात्मको वा॥ (A.H.Ni.10/40)

◉ दृष्ट्वा प्रमेहं मधुरं सपिच्छं मधूपमं स्याद्द्विविधो विचारः।
क्षीणेषु दोषेष्वनिलात्मकः स्यात् सन्तर्पणाद्वा कफसम्भवः स्यात्॥ (Ca.Ci.6/55)

Ācārya Vāgbhaṭa while describing the prognosis, he classified Prameha into two types, i.e. **Santarpaṇa janya** and **Apatarpaṇa janya.** Santarpaṇa janya Prameha is due to Kapha and Apatarpaṇa janya Prameha is due to Vāta. Even Ācārya Caraka describes the same.

◉ .....जायते स किल द्विधा॥ क्रुद्धे धातुक्षयाद्वायौ दोषावृतपथेऽथवा। आवृतो दोषलिङ्गानि सोऽनिमित्तं प्रदर्शयेत्॥ क्षीणः क्षणात्क्षणात् पूर्णो भजते कृच्छ्रसाध्यताम्। (A.H.Ni.10/18-20)

Again Ācārya Vāgbhaṭa classified Prameha into two types based on the aggravation of Vāta caused by Dhātukṣaya (depletion of tissues) and Doṣāvṛta (obstruction of the passage of Vāta by the doṣas). In Avṛtajanya Prameha, the feature of Doṣa which covers the Vāta is manifested without any specific reason. In this type, the basti (bladder) gets Kṣīṇaḥ kṣaṇātkṣaṇāt pūrṇobhajate (sometimes empty and sometimes filled); hence it is considered as Kṛcchra sādhya (difficult to cure)

Even Ācārya Caraka classified Prameha into two types based on the Saṃprāpti i.e. Āvaraṇajanya and Dhatukṣayajanya Prameha in Sūtra sthāna. Āvāraṇajanya Prameha occurs due to the Mārgāvaraṇa caused by Kapha and Medas and Dhatukṣaya is due to vitiation of Vāta. This differentiation is also important as the treatment differs in these two distinct Saṃprāpti of the Prameha.

◉ द्वौ प्रमेहौ भवतः- सहजोऽपथ्यनिमित्तश्च । तत्र सहजो मातृपितृबीजदोषकृतः, अहिताहारजोऽपथ्यनिमित्तः । तयोः पूर्वेणोपद्रुतः कृशो रूक्षोऽल्पाशी पिपासुर्भृशं परिसरणशीलश्च भवति; उत्तरेण स्थूलो बह्वाशी स्निग्धः शय्यासनस्वप्नशीलः प्रायेणेति ॥ (Su.Ci.11/3)

Prameha is classified into two types based on the etiological factors i.e Sahaja and Apathyanimittaja.

◈ **Sahaja Pramehi** is Kṛśa (emaciated), Rūkṣa (dryness of the body), Alpāśī (intake less food), Pipāsurbhṛśaṃ (has excessive thirst) and Parisaraṇaśīla (has restless disposition) as a result of Mātṛpitṛbījadoṣa (defect in the germinal seeds of parents) (Ācārya Caraka described it as Jātaḥ Prameha);

◈ **Apathyanimittaja Pramehi** is Sthūla (obese), Bahvāśī (intake more food), Snigdha (has unctuousness of the body) and Śayyāsanasvapnaśīla (has fondness for sedentary habits like comfortable lying or sitting) as a result of Ahitāhāra (intake of unwholesome food).

◉ प्रकृतिप्रभवश्चैव नरस्य स्वकृतस्तथा । ज्ञेयः प्रमेहो द्विविधस्तस्य वक्ष्यामि लक्षणम् ॥ (Bhe.Sa.Ni.6/1)

Ācārya Bhela classified Prameha into two types i.e. Prakṛtija (Sahaja meha) and Svakṛtaja Prameha (Janmottaraja meha).

◉ जलप्रमेहो रुधिरप्रमेहः पूयमेहो लवणमेहः। तक्रप्रमेहः खटिकाप्रमेहः शुक्रप्रमेहः कथित पुरस्तात्॥ स्याच्छर्करामेहो वसाप्रमेहो रसप्रमेहोऽन्यघृतप्रमेहः। पित्तप्रमेही कफप्रमेहिनश्च मधुप्रमेहीति विभावयेच्च॥ (Hā.Sa.Tṛ.28/3-4)

Ācārya Hārīta classified Prameha into fourteen types with nomenclature different from other Ācāryās. They are Jalameha, Rudhirameha, Pūyameha, Lavaṇameha, Takrameha, Khaṭikāmeha, Śukrameha, Śarkarāmeha, Vasāmeha, Rasameha, Ghṛtameha, Pittameha, Kaphameha and Madhumeha.

- ◉ सपूर्वरूपाः कफपित्तमेहाः क्रमेण ये वातकृताश्च मेहाः। साध्या न ते, पित्तकृतास्तु याप्याः, साध्यास्तु मेदो यदि न प्रदुष्टम्॥ (Ca.Ci.6/46)

Ācārya Caraka while describing the prognosis, he had classified Prameha into two types based on the premonitory features i.e. Pūrvarūpa Sahitā Prameha (Prameha manifested with premonitory features) and Pūrvarūpa Rahitā Prameha (Prameha manifested without premonitory features). In this verse, he had mentioned that the Kaphaja and Pittaja Pramehās preceded with their premonitory features are incurable. Vātaja Prameha described in order is incurable (Cakrapāṇi opines that the Vātaja Pramehās are incurable even if manifested without any premonitory features). Though, Pittaja Prameha is Yāpya (palliable), it becomes Sādhya (curable) if it is manifested without vitiation of Medas.

Sāmānya Nidāna and Saṃprāpti

- ◉ आस्यासुखं स्वप्नसुखं दधीनि ग्राम्यौदकानूपरसाः पयांसि। नवान्नपानं गुडवैकृतं च प्रमेहहेतुः कफकृच्च सर्वम्॥ (Ca.Ci.6/4)

Āsyāsukhaṃ (pleasure of sedentary habits like comfortable sitting), Svapnasukhaṃ (pleasure of comfortable sleeping), Dadhīni (excessive intake of curds), Grāmyaudakānūpa rasa (meat soup of domestic, aquatic and marshy animals), Payāṃsi (milk), Navānnapānaṃ (diet and drinks prepared from recently harvested cereals), Guḍavaikṛta (preparations made with jaggery) and Sarva Kaphakṛcca (all Kapha aggravating factors) are causative factors of Prameha.

- ◉ मेदश्च मांसं च शरीरजं च क्लेदं कफो बस्तिगतं प्रदूष्य। करोति मेहान् समुदीर्णमुष्णैस्तानेव पित्तं परिदूष्य चापि॥ क्षीणेषु दोषेष्ववकृष्य बस्तौ धातून् प्रमेहाननिलः करोति। दोषो हि बस्तिं समुपेत्य मूत्रं सन्दूष्य मेहाञ्जनयेद्यथास्वम्॥ (Ca.Ci.6/5-6)

The aggravated Kapha vitiates the Medas (fat), Māṃsa (muscle) and Kleda (moistureness) of the body located in Basti (urinary bladder) and produces Kaphaja Meha. In the same manner, the Pitta gets aggravated due to intake of Uṣṇa (hot substances) and produces Pittaja Meha by affecting the aforesaid entities. When the Pitta and Kapha are in decreased state, the Vāta gets aggravated and pulls the dhātus (Majjā, Ojas and Lasīkā) to the Basti (urinary bladder) and produces Vātaja Prameha. The doṣas reaching the Basti (urinary bladder) pollutes the Mūtra (urine) and produces respective types of Prameha.

- ◉ दिवास्वप्नाव्यायामालस्यप्रसक्तं शीतस्निग्धमधुरमेद्यद्रवान्नपानसेविनं पुरुषं जानियात् प्रमेही भविष्यतीति। तस्य चैवम्प्रवृत्तस्यापरिपक्वा एव वातपित्तश्लेष्माणो यदा मेदसा सहैकत्वमुपेत्य मूत्रवाहिस्रोतांस्यनुसृत्याधो गत्वा बस्तेर्मुखमाश्रित्य निर्भिद्यन्ते तदा प्रमेहाञ्जनयन्ति ॥ (Su.Ni.6/3-4)

Ācārya Suśruta described that the person who indulges Divāsvapna (day sleeping), Avyāyāma (absence of physical exercise), Ālasyaprasakta (constant laziness) and Śitasnigdhamadhuramedya dravānnapānasevinaṃ (intake of cold, unctuous, sweet, fatty and watery foods and drinks), the Aparipakvā Vātādi doṣas (immature Vāta, Pitta and Kapha) along with Medas moves downwards in the Mūtravaha srotas and gets located in the Bastimukha (opening aperture of the bladder) are excreted thus produces Prameha.

Viśeṣa Nidāna and Saṃprāpti

Kaphaja Prameha

- ◉ हायनकयवकचीनकोद्दालकनैषधेत्कटमुकुन्दकमहाव्रीहिप्रमोदकसुगन्धकानां नवानामतिवेलमतिप्रमाणेन चोपयोगः, तथा सर्पिष्मतां नवहरेणुमाषसूप्यानां, ग्राम्यानूपौदकानां च मांसानां, शाकतिलपललपिष्टान्न पायसकृशराविलेपीक्षुविकाराणां, क्षीरनवमद्यमन्दकदधिद्रवमधुरतरुणप्रायाणां चोपयोगः, मृजाव्यायाम वर्जनं, स्वप्नशयनासनप्रसङ्गः, यश्च कश्चिद्विधिरन्योऽपि श्लेष्ममेदोमूत्रसञ्जननः, स सर्वो निदानविशेषः॥ बहुद्रवः श्लेष्मा दोषविशेषः॥ बह्वबद्धं मेदो मांसं शरीरजक्लेदः शुक्रं शोणितं वसा मज्जा लसीका रसश्चौजःसङ्ख्यात इति दूष्यविशेषाः॥ (Ca.Ni.4/5-7)

Due to frequent and excessive intake of newly harvested cereals like Hāyanaka, Yavaka, Cīnaka, Uddālaka, Naiṣadha, Itkaṭa, Mukundaka, Mahāvrīhi, Pramodaka and Sugandhaka, and also use of Sarpiṣmatāṃ navahareṇumāṣasūpyānāṃ (soups prepared from fresh legumes like Hareṇu and Māṣa along with ghee), Grāmyānūpaudakānāṃ māṃsānāṃ (meat of domestic, marshy and aquatic animals), Śāka (vegetables), Tila (sesame), Palala (oil cake prepared from Tila), Piṣṭānna (pastries), Pāyasa (rice cooked with milk and sweetened), Kṛśara (dish prepared from sesamum and grains), Vilepī (thick rice gruel), Ikṣuvikārāṇāṃ (preparations of sugarcane), Kṣīra (milk), Navamadya (fresh wine), Mandakadadhi (less curdledcurd), Dravamadhurataruṇa prāyāṇāṃ (use of liquid, sweet and fresh substances), Mṛjāvyāyāmavarjana (avoiding purificatory therapies and exercise) and Svapnaśayanāsanaprasaṅga (excessive indulgence of sleep, lying down and sitting) are the causative factors of Kaphaja Prameha which leads to aggravation of Kapha, Medas and Mūtra.

This Kapha (Bahudravaḥ Śleṣma - especially liquid portion of Kapha) along with Bahvabaddha (abundant and non-compact) Medas, Māṃsa, Kleda, Śukra, Śoṇita, Vasā, Majjā, Lasīkā, Rasa and Ojas, converts the Dravadhātus into Mūtra and also blocks the Mūtravaha sotas originating from Vṛkka and Basti, which are already afflicted with Medas and Kleda resulting in ten types of Kaphaja Prameha which is Sādhya (curable) due to Samānaguṇamedaḥsthānakatvāt (located in the Medas having similar properties to those of Kapha), Kaphasya pradhānyāt (predominance of Kapha) and Samakriyatvāt (similarity in the treatment measures for both doṣa and dūṣyās).

Pittaja Prameha Nidāna and Saṃprāpti

- उष्णाम्ललवणक्षारकटुकाजीर्णभोजनोपसेविनस्तथाऽतितीक्ष्णातपाग्निसन्तापश्रम क्रोध विषमाहारोपसेविनश्च तथाविधशरीरस्यैव क्षिप्रं पित्तं प्रकोपमापद्यते, तत्तु प्रकुपितं तयैवानुपूर्व्या प्रमेहानिमान् षट् क्षिप्रतरमभिनिर्वर्तयति॥ (Ca.Ni.4/24)

Excessive intake of Uṣṇāmlalavaṇakṣārakaṭu (hot, sour, saline, alkaline and pungent food substances), Ajīrṇabhojana (intake of food during indigestion), Atitīkṣṇātapāgnisantāpa (exposure to intense heat of hot sun and fire), Śrama (physical exertion), Krodha (anger), and Viṣamāhāropasevina (intake of contrary food substances) are the causative factors of Pittaja Prameha which leads to aggravation of Pitta immediately and manifests six types of Pittaja Prameha which is Yāpya (palliable) due to Saṃsṛṣṭadoṣa (combination of doṣas), Medaḥsthāna (located in the Medas) and Viruddhopakrama (contrariety of treatment).

Vātaja Prameha Nidāna and Saṃprāpti

- कषायकटुतिक्तरूक्षलघुशीतव्यवायव्यायामवमनविरेचनास्थापनशिरोविरेचनातियोगसन्धारणानशनाभिघातातपोद्वेगशोकशोणितातिषेकजागरणविषमशरीरन्यासानुपसेवमानस्य तथाविधशरीरस्यैव क्षिप्रं वातः प्रकोपमापद्यते॥ (Ca.Ni.4/36)

Excessive intake of Kaṣāyakaṭutiktarūkṣalaghuśīta (astringent, pungent, bitter, rough, light and cold food substances), Vyavāyavyāyāma (excessive indulgences in sexual intercourse and exercise), Vamanavirecanāsthāpanaśirovirecanātiyogāt (excessive use of emesis, purgation, non-unctuous enema and elimination of doṣas from the head), Sandhāraṇā (suppressing natural urges), Anaśana (fasting), Abhighāta (injury), Atāpa (exposure to sun), Udvega (anxiety), Śoka (grief), Śoṇitātiṣeka (excessive blood-letting), Jāgaraṇa (awakening at night) and Viṣamaśarīranyāsānupasevamāna (irregular posture of the body) are the causative factors of Vātaja Prameha which leads to aggravation of Vāta immediately and manifests four types of Vātaja Prameha which is Asādhya (incurable) due to Mahātyayikatvāt (their severity) and Viruddhopakrama (contrariety of treatment)

Saṃprāpti Ghaṭaka

Doṣa	Śleṣma Pradhāna Tridoṣas
Dūṣya	Rasa, Rakta, Māṃsa, Meda, Majjā, Śukra, Vasā, Ojas, Lasikā and Kleda.
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhya janya Āma
Srotas	Meda, Māṃsa, Udaka, Rasa, Rakta, Majjā, Śukra and Mūtra vaha
Srotoduṣṭi	Saṅga, Vimārga gamana and Atipravṛtti
Sañcāra sthāna	Sarva Śarīra
Adhiṣṭhāna	Basti
Udbhava sthāna	Āmāśaya
Vyakta sthāna	Basti and Sarva Śarīra
Rogamārga	Madhyama

Pūrvarūpa of Prameha

- स्वेदोऽङ्गगन्धः शिथिलाङ्गता च शय्यासनस्वप्नसुखे रतिश्च। हृन्नेत्रजिह्वाश्रवणोपदेहो घनाङ्गता केशनखातिवृद्धिः॥ शीतप्रियत्वं गलतालुशोषो माधुर्यमास्ये करपाददाहः। भविष्यतो मेहगदस्य रूपं मूत्रेऽभिधावन्ति पिपीलिकाश्च॥ (Ca.Ci.6/13-14)

Sveda (perspiration), Aṅgagandha (foul smell in the body), Śithilāṅgatā (looseness of the body), Śayyāsanasvapnasukhe ratiśca (delight in comfortable lying, sitting and sleeping), Hṛnnetrajihvāśravaṇopadeho (feeling as if cardiac region, eyes, tongue and ears are smeared), Ghanāṅgatā (heaviness of body parts), Keśanakhātivṛddhi (excessive growth of hairs and nails), Śītapriyatva (desire for cold things), Galatāluśoṣa (dryness of throat and palate), Mādhuryamāsye (sweet taste in the mouth) and Karapādadāha (burning sensation in palms and soles) are the prodromal features of Prameha. Besides these, Mūtreabhidhāvanti pipīlikāśca (ants rush towards the urine).

- तेषां तु पूर्वरूपाणि- हस्तपादतलदाहः स्निग्धपिच्छिलगुरुता गात्राणां मधुरशुक्लमूत्रता तन्द्रा सादः पिपासा दुर्गन्धश्च श्वासस्तालुगलजिह्वादन्तेषु मलोत्पत्तिर्जटिलीभावः केशानां वृद्धिश्च नखानाम् ॥ (Su.Ni.6/5)

Ācārya Suśruta added Snigdhapicchilagurutā gātraṇāṃ (unctuousness, sliminess and heaviness of the body), Madhuraśuklamūtratā (sweetness and whitishness of urine), Tandrā (drowsiness), Sāda (lethargy), Pipāsā (thirst), Durgandha (foul smell), Śvāsa (dyspnoea), Tālugalajihvādanteṣu malotpatti (production of dirtiness in the palate, throat, tongue and teeth), Jaṭilībhāva keśānāṃ (matting together of hairs) and Vṛddhiśca nākhānām (increased growth of nails).

- प्रमेहपूर्वरूपाणामाकृतिर्यत्र दृश्यते । किञ्चिच्चाप्यधिकं मूत्रं तं प्रमेहिणमादिशेत् ॥
  कृत्स्नान्यर्धानि वा यस्मिन् पूर्वरूपाणि मानवे । प्रवृत्तमूत्रमत्यर्थं तं प्रमेहिणमादिरोत् ॥ (Su.Ni.6/22-23)

Here, Ācārya Suśruta described the significance of Pūrvarūpa i.e. if all the Pūrvarūpa are clearly exhibited along with Āpyadhikamūtra (a slight increase in urine) then the patient should be considered to be a Pramehi. Even if half of the Pūrvarūpa are clearly exhibited along with Atyarthamūtrapravṛtti (excessive urination) then the patient should be considered to be a Pramehi.

Sāmānya Lakṣaṇās of Prameha

- ◉ तत्राविलप्रभूतमूत्रलक्षणाः सर्व एव प्रमेहा भवन्ति ॥ (Su.Ni.6/6)
- ◉ सामान्यं लक्षणं तेषां प्रभूताविलमूत्रता। (A.H.Ni.10/7)
- ◉ प्रभूतेत्यादि प्रभूतमूत्रत्वं दूष्यद्रवधातुसम्बन्धात् आविलत्वं दोषदूष्यसंसर्गात्। (Ma.M.N.33/6)

Āvilamūtrata (turbid urination) and Prabhūtamūtratā (excessive micturition) are manifested as symptoms in all types of Prameha. Ācārya Madhukośa, while commenting on this mentioned that Prabhūtamūtratā (excessive micturition) is related to Dravadhātu dūṣya (corruption of watery tissues of the body) and Āvilamūtrata (turbid urination) is due to Doṣadūṣyasaṃsargāt (combination of Doṣa and Dūṣya).

Viśeṣa Lakṣaṇās of Prameha

Kaphaja Prameha Lakṣaṇās

◉ अच्छं बहु सितं शीतं निर्गन्धमुदकोपमम्। श्लेष्मकोपान्नरो मूत्रमुदमेही प्रमेहति॥
अत्यर्थमधुरं शीतमीषत्पिच्छिलमाविलम्। काण्डेक्षुरसमङ्काशं श्लेष्मकोपात् प्रमेहति॥
यस्य पर्युषितं मूत्रं सान्द्रीभवति भाजने। पुरुषं कफकोपेन तमाहुः सान्द्रमेहिनम्॥
यस्य संहन्यते मूत्रं किञ्चित् किञ्चित् प्रसीदति। सान्द्रप्रसादमेहीति तमाहुः श्लेष्मकोपतः॥
शुक्लं पिष्टनिभं मूत्रमभीक्ष्णं यः प्रमेहति। पुरुषं कफकोपेन तमाहुः शुक्लमेहिनम्॥
शुक्राभं शुक्रमिश्रं वा मुहुर्मेहति यो नरः। शुक्रमेहिनमाहुस्तं पुरुषं श्लेष्मकोपतः।
अत्यर्थमधुरं शीतं मूत्रं मेहति यो भृशम्। शीतमेहिनमाहुस्तं पुरुषं श्लेष्मकोपतः॥
मूर्तान्मूत्रगतान् दोषानणून्मेहति यो नरः। सिकतामेहिनं विद्यात्तं नरं श्लेष्मकोपतः॥
मन्दं मन्दमवेगं तु कृच्छ्रं यो मूत्रयेच्छनैः। शनैर्मेहिनमाहुस्तं पुरुषं श्लेष्मकोपतः॥
तन्तुबद्धमिवालालं पिच्छिलं यः प्रमेहति। आलालमेहिनं विद्यात्तं नरं श्लेष्मकोपतः॥ (Ca.Ni.4/13-22)

- ◈ **Udakameha:** in this, one excretes urine which is Accha (transparent), Bahu (excessive in amount), Sita (white), Śīta (cold), Nirgandha (odorless) and Udakopamam (it resembles water).
- ◈ **Ikṣuvālikārasameha:** Atyartha madhura (excessively sweet), Śīta (cold), Iṣatpicchilam (slight slimy), Avilam (turbid) and Kāṇḍekṣurasasaṅkāśaṃ (it resembles the juice of Kāṇḍekṣu).
- ◈ **Sāndrameha:** Paryuṣitaṃ mūtraṃ sāndrībhavati bhājane (viscosity of the urine increased when kept in a pot overnight).
- ◈ **Sāndraprasādameha:** Saṃhanyate mūtraṃ kiñcit kiñcit prasīdati (when the urine is kept in a pot overnight, it becomes partially viscous and partially clear).
- ◈ **Śuklameha:** Śukla (white in colour), Piṣṭanibhaṃ (it resembles paste of flour) and Mūtrabhīkṣṇaṃ (passes urine frequently).

- ◈ **Śukrameha:** Śukrābhaṃ (resembles the semen), Śukramiśraṃ (urine excreted mixed with semen) and Muhurmehati (passes urine frequently).
- ◈ **Śītameha:** Atyartha madhura (excessively sweet), Śīta (cold) and Mehati yo bhṛśaṃ (passes urine frequently or excessively)
- ◈ **Sikatāmeha:** Mūrtānmūtragatān doṣānanūmehati (the vitiated doṣas passed with the urine in the form of Kaṭhina (hard small particles))
- ◈ **Śanairmeha:** Mandaṃ (small quantity of urine), Mandamavegaṃ (passed very slowly), Kṛcchraṃ yo mūtrayecchanaiḥ (with difficulty).
- ◈ **Ālālameha:** Tantubaddhaivā (urine appears like thread), Picchilaṃ (slimy) and Ālālaṃ (it resembles saliva).
- ◈ **Surāmeha:** Surātulyaṃ (urine excretes similar to wine)
- ◈ **Lavaṇameha:** Viśadaṃ (the excreted urine is clear) and Lavaṇatulyaṃ (it is similar to saline water)
- ◈ **Piṣṭameha:** Hṛṣṭaromā (horripilation) and Piṣṭarasatulyaṃ (it is similar to water mixed with pasted flour)
- ◈ **Phenameha:** Stokaṃ stokaṃ saphenamacchaṃ (clear and frothy urine passed in small quantities repeatedly).

Pittaja Prameha Lakṣaṇās

◉ गन्धवर्णरसस्पर्शैर्यथा क्षारस्तथाविधम्। पित्तकोपान्नरो मूत्रं क्षारमेही प्रमेहति॥ मसीवर्णमजस्रं यो मूत्रमुष्णं प्रमेहति। पित्तस्य परिकोपेण तं विद्यात् कालमेहिनम्॥ चाषपक्षनिभं मूत्रमम्लं मेहति यो नरः। पित्तस्य परिकोपेण तं विद्यान्नीलमेहिनम्॥ विस्रं लवणमुष्णं च रक्तं मेहति यो नरः। पित्तस्य परिकोपेण तं विद्याद्रक्तमेहिनम्॥ मञ्जिष्ठोदकसङ्काशं भृशं विस्रं प्रमेहति। पित्तस्य परिकोपात्तं विद्यान्माञ्जिष्ठमेहिनम्॥ हरिद्रोदकसङ्काशं कटुकं यः प्रमेहति। पित्तस्य परिकोपात्तं विद्याद्धारिद्रमेहिनम्॥ (Ca.Ni.4/29-34)

- ◈ **Kṣārameha:** in this, the urine is similar to Kṣāra (alkali) in Gandhavarṇarasasparṣa (smell, colour, taste and touch).
- ◈ **Kālameha:** Masīvarṇa (passes ink coloured), Ajasraṃ (perpetually or constant) and Uṣṇamūtra (hot urine).
- ◈ **Nīlameha:** Amlamūtra (passes sour urine) and Cāṣapakṣanibhaṃ (it resembles the colour of cāṣa bird feather).
- ◈ **Raktameha:** Lavaṇamuṣṇaṃcaraktaṃ (passes urine as saline, hot and red in colour) and Visraṃ (has smell like that of raw meat)
- ◈ **Mañjiṣṭhameha:** Mañjiṣṭhodakasaṅkāśaṃ bhṛśaṃ (passes excessive urine resembling the colour of decoction of Mañjiṣṭha) and Visraṃ (has smell like that of raw meat).

◈ **Haridrāmeha:** Kaṭu mūtra (passes pungent urine) and has Haridrodakasaṅkāśaṃ (the colour of decoction of Haridrā).

◈ **Amlameha:** Amlarasagandha (the urine is with sour taste and smell)

Vātaja Prameha Lakṣaṇās

◉ वसामिश्रं वसाभं वा मुहुर्मेहति यो नरः। वसामेहिनमाहुस्तमसाध्यं वातकोपतः॥
मज्जानं सह मूत्रेण मुहुर्मेहति यो नरः। मज्जमेहिनमाहुस्तमसाध्यं वातकोपतः॥
हस्ती मत्त इवाजस्रं मूत्रं क्षरति यो भृशम्। हस्तिमेहिनमाहुस्तमसाध्यं वातकोपतः॥
कषायमधुरं पाण्डु रूक्षं मेहति यो नरः। वातकोपादसाध्यं तं प्रतीयान्मधुमेहिनम्॥ (Ca.Ni.4/41-44)

◈ **Vasāmeha:** Vasāmiśraṃ (the urine excreted along with fat), Vasābhaṃ vā (or similar to fat) and Muhurmehati (passes frequently) and it is Asādhya (incurable).

◈ **Majjāmeha:** Majjānaṃ (the urine excreted along with Majjā) and it is Asādhya (incurable).

◈ **Sarpimeha:** Sarpiḥprakāśaṃ (urine excreted is similar to ghee).

◈ **Hastimeha:** Hastīmatta ivājasraṃ mūtraṃ kṣarati yo bhṛśaṃ (perpetually or constantly passing urine in an excessive quantity like an intoxicated elephant) and it is Asādhya (incurable).

◈ **Madhumeha/Kṣaudrameha:** Kaṣāyamadhuraṃ (passes astringent and sweet urine), Pāṇḍu (pale in colour) and Rūkṣaṃ (non-unctuous). It resembles Kṣaudrarasavarṇa (the colour of honey) and it is Asādhya (incurable).

◉ कालेनोपेक्षिताः सर्वे यद्यान्ति मधुमेहताम्॥ मधुरं यच्च सर्वेषु प्रायो मध्विव मेहति।
सर्वेऽपि मधुमेहाख्या माधुर्याच्च तनोरतः॥ (A.H.Ni.10/20-21)

Ācārya Vāgbhaṭa described that, all the Prameha left untreated becomes Madhumeha in course of time. All those in which the urine becomes Madhura rasa (sweet in taste) and it resembles Madhviva (honey in colour) and the body also becomes Mādhuryā (sweet) are called Madhumeha.

Prameha and Madhumeha

There is lot of controversy regarding the word Prameha and Madhumeha. Some opines Madhumeha is a particular type of Prameha and others opines that Madhumeha is the last stage of Prameha. Even though the term Madhumeha is a type of Prameha, it is used as synonym for Prameha in the following circumstances.

◉ माधुमेहिका इत्यत्र मधुमेहशब्दः सामान्येन प्रमेहवचनः (Ca.Ca.Sū.17/3-7)

But Ācārya Cakrapāṇi described that the word Madhumeha refers to Prameha in general, not any specific types.

◉ विना प्रमेहमप्येता जायन्ते दुष्टमेदसः। (Ca.Sū.17/104)

The Piḍaka appears due to vitiation of Medas even without Prameha. If the term Madhumeha were to denote only a particular type of Prameha which normally causes Piḍaka, then the appropriate term to be used in this verse should have been Madhumeha and not Prameha.

◉ प्रमेहिणां याः पिडका मयोक्ता रोगाधिकारे पृथगेव सप्त। ताः शल्यविद्भिः कुशलैश्चिकित्स्याः शस्त्रेण संशोधनरोपणैश्च॥ (Ca.Ci.6/58)

◉ प्रमेहिणां याः पिडका मयोक्ता रोगाधिकारे पृथगेव सप्त इत्यादि। तथा मधुमेहपिडकानां चिकित्सितोपदेशाच्च सर्वमेहभवत्वं पिडकानां, मधुमेहभवत्वे हि मधुमेहस्यैवासाध्यत्वान्न तद्भवपिडकानामुपक्रमणीयत्वमस्ति; किञ्च प्रदेशान्तरेऽपि मधुमेहशब्देनायं सर्वप्रमेहानुक्तवान् । (Ca.Ca.Sū.17/3-7)

The seven types of Piḍaka developing in the Pramehi are discussed separately in the Rogādhikara adhyāya and the treatment of Piḍaka arising out of Madhumeha has also been described. The Piḍaka arising out of Madhumeha is Asādhya (incurable), since Madhumeha itself is Asādhya (incurable). This shows the word Madhumeha occuring in the present context stands for Prameha.

◉ गुल्मी च मधुमेही च राजयक्ष्मी च यो नरः॥ अचिकित्स्या भवन्त्येते बलमांसक्षये सति। (Ca.I.9/8-9)

In Indriya sthāna, it is described that those suffering from Gulma, Madhumeha and Rājayakṣma become incurable due to Balamāṃsakṣaya (loss of strength and muscles). In this context, if the word Madhumeha denotes Vātaja type of Prameha, then mentioning Balamāṃsakṣaya (loss of strength and muscles) is unnecessary because the disease being incurable itself.

◉ तस्मान्मधुमेहशब्दः सर्वप्रमेहे मधुमेहविशेषे च वर्तते; यथा तृणशब्दः सर्वतृणे तृणविशेषे च वर्तते। (Ca.Ca.Ci.6/57)

Acārya Cakrapāṇi while commenting the Jātaḥ Pramehi or Madhumehi, he had mentioned that the word Tṛṇa (grass) refers to Tṛṇa in general and also specific similarly, the word Madhumeha denotes all Pramehās specifically to Madhumeha.

Prameha Piḍakā

◉ उपेक्षयाऽस्य जायन्ते पिडकाः सप्त दारुणाः। मांसलेष्ववकाशेषु मर्मस्वपि च सन्धिषु॥
शराविका कच्छपिका जालिनी सर्षपी तथा। अलजी विनताख्या च विद्रधी चेति सप्तमी॥ (Ca.Sū.17/82-83)

If the disease Prameha is neglected, then it causes seven types Dāruṇa piḍakā (serious types of boils) in the Māṃsa (muscular areas), Marma (vital parts) and Sandhi (joints) of the body. They are Śarāvikā, Kacchapikā, Jālinī, Sarṣapī, Alajī, Vinatā and Vidradhī.

◉ तत्र वसामेदोभ्यामभिपन्नशरीरस्य त्रिभिर्दोषैश्चानुगतधातोः प्रमेहिणो दश पिडका जायन्ते । तद्यथा- शराविका, सर्षपिका, कच्छपिका, जालिनी, विनता, पुत्रिणी, मसूरिका, अलजी, विदारिका, विद्रधिका चेति ॥ (Su.Ni.6/14)

The aggravated Tridoṣas after spreading in the dhātus (tissues) with Vasā and Medas in the body of Pramehi produces ten types of Piḍakās (boils). They are Śarāvikā, Sarṣapikā, Kacchapikā, Jālinī, Vinatā, Putruṇī, Masūrikā, Alajī, Vidārikā and Vidradhikā.

- **Śarāvikā:** Śarāvamātra tadrūpa nimnamadyā (has the appearance and size of earthen saucer with depression in the centre)
- **Sarṣapikā:** Gaurasarṣapasaṃsthānā tatpramāṇā (has the shape and size of white mustard)
- **Kacchapikā:** Sadāha Kūrmasaṃsthāna (has the shape of tortoise associated with burning sensation)
- **Jālinī:** Tīvradāhā tu māṃsajālasamāvṛtā (surrounded by a network of muscles with severe burning sensation)
- **Vinatā:** Mahatī piḍakā nīla piḍakā (big and blue boil)
- **Putruṇī:** Mahatyalpācitā piḍakā (big boil surrounded by multiple small boils)
- **Masūrikā:** Masūrasaṃsthānā (has the shape and size of lentils)
- **Alajī:** Raktā sitā sphoṭavatī dāruṇā (reddish white indurated blisters)
- **Vidārikā:** Vidārīkandavadvṛttā kaṭhinā (round and hard like a gourd)
- **Vidradhikā:** Vidradherlakṣaṇairyuktā (boil associated with the features of Vidradhi)

Sāmānya Upadravā of Prameha

- उपद्रवास्तु खलु प्रमेहिणां तृष्णातीसारज्वरदाहदौर्बल्यारोचकाविपाकाः पूतिमांसपिडकालजीविद्रध्यादयश्च तत्प्रसङ्गाद्भवन्ति॥ (Ca.Ni.4/48)

Tṛṣṇā (thirst), Atīsāra (diarrhoea), Jvara (fever), Dāha (burning sensation), Daurbalya (weakness), Arocakā (anorexia), Avipākā (indigestion), Pūtimāṃsapiḍaka (boils that putrefies the muscles), Alajī, Vidradhi (abscess) etc. diseases develop as a complication of Prameha.

Viśeṣa Upadravā of Prameha

Kaphaja Prameha Upadravā

- मक्षिकोपसर्पणमालस्यं मांसोपचयः प्रतिश्यायः शैथिल्यारोचकाविपाकाः कफप्रसेकच्छर्दिनिद्राकासश्वासाश्चेति श्लेष्मजानामुपद्रवाः ॥ (Su.Ni.6/13)

Makṣikopasarpaṇa (flies attracted towards the urine), Ālasaya (lassitude), Māṃsopacaya (growth of muscles), Pratiśyāya (corrhyza), Śaithilya (flaccidity of the limbs), Arocaka (anorexia), Avipāka (indigestion), Kaphapraseka (mucous discharge), Chardi (vomiting), Nidrā (excessive sleep), Kāsa (cough) and Śvāsa (dyspnoea) are the complications of Kaphaja Prameha.

Pittaja Prameha Upadravā

- वृषणयोरवदरणं बस्तिभेदो मेढ्रतोदो हृदि शूलमम्लीकाज्वरातीसारारोचका वमथुः परिधूपनं दाहो मूर्च्छा पिपासा निद्रानाशः पाण्डुरोगः पीतविण्मूत्रनेत्रत्वं चेति पैत्तिकानां; ॥ (Su.Ni.6/13)

Vṛṣaṇayoravadaraṇaṃ (bursting type of sensation in the testes), Bastibheda (splitting type of pain in the bladder), Meḍhratoda (pricking pain in the penis), Hṛdiśūla (pain in the heart), Amlikā (sour eructations), Jvara (fever), Atīsāra (diarrhoea), Arocakā (anorexia), Vamathu (vomiting), Paridhūpanaṃ (feeling of emission of smoke from the entire body), Dāha (burning sensation), Mūrcchā (fainting), Pipāsā (thirst), Nidrānāśa (loss of sleep), Pāṇḍuroga (anameia), and Pītaviṇmūtranetratvaṃ (yellowish discoloration of faeces, urine and eyes) are the complications of Pittaja Prameha.

Vātaja Prameha Upadravā

- हृद्ग्रहो लौल्यमनिद्रा स्तम्भः कम्पः शूलं बद्धपुरीषत्वं चेति वातजानाम् । (Su.Ni.6/13)

Hṛdgraha (contracting sensation in the heart), Laulya (craving for various tastes), Anidra (insomnia), Stambha (stiffness), Kampa (tremors), Śūla (colic) and Baddhapurīṣatva (constipation of the bowels) are the complications of Vātaja Prameha.

Sādhyāsādhyata of Prameha

- सपूर्वरूपाः कफपित्तमेहाः क्रमेण ये वातकृताश्च मेहाः।
  साध्या न ते, पित्तकृतास्तु याप्याः, साध्यास्तु मेदो यदि न प्रदुष्टम्॥ (Ca.Ci.6/56)

Kaphaja and Pittaja Pramehās preceded with their premonitory features are incurable. Vātaja Prameha described in order is incurable (Cakrapāṇi opines that the Vātaja Pramehās are incurable even if manifested without any premonitory features). Though, Pittaja Prameha is Yāpya (palliable), but it becomes Sādhya (curable) if it is manifested without vitiation of Medas.

- जातः प्रमेही मधुमेहिनो वा न साध्य उक्तः स हि बीजदोषात्।
  ये चापि केचित् कुलजा विकारा भवन्ति तांश्च प्रवदन्त्यसाध्यान्॥ (Ca.Ci.6/57)

The person born with the disease Prameha or Madhumeha is Asādhya (incurable), because this disease manifests due to Bījadoṣāt (defect of germinal seeds of parents). Similarly, other Kulaja Vikāras (hereditary diseases) are considered as incurable.

- गुदे हृदि शिरस्यंसे पृष्ठे मर्मणि चोत्थिताः । सोपद्रवा दुर्बलाग्नेः पिडकाः परिवर्जयेत् ॥
  कृत्स्नं शरीरं निष्पीड्य मेदोमज्जवसायुतः । अधः प्रक्रमते वायुस्तेनासाध्यास्तु वातजाः ॥ (Su.Ni.6/20-21)

Piḍakā arising in the Guda (anus), Hṛdi (cardiac region), Śiras (head), Aṃsa (shoulder), Pṛṣṭha (back) and Marma (vital parts of the body) associated with Upadravā (complications) in a person with Durbalāgne (weak digestive fire) should be discarded from the treatment as it is incurable. The Vāta along with Medas, Majjā and Vasā compresses the lower part of the body; hence the Vātaja Prameha (or boils of Vāta origin) is Asādhya (incurable).

In Contemporary science,

Prameha is a group of urinary disorders characterized by excessive urination. For clinical discussion it can be predominantly correlated with conditions like Diabetes Mellitus in contemporary science. (Please refer Topic 39).

Short Answer Questions

1. Explain Sāmānya Nidāna and Saṃprāpti of Prameha
2. Describe Pūrvarūpa of Prameha
3. Explain the types of Prameha and its lakṣaṇās
4. Explain Prameha Piḍakā
5. Describe Sāmānya and Viśeṣa Upadrava of Prameha
6. Describe Sādhyāsādhyata of Prameha

Long Answer Questions

1. Define Prameha, and explain the Nidāna, Saṃprāpti, Bheda and Lakṣaṇās of Prameha.

◆

TOPIC 39

Diabetes Mellitus and Pancreatitis

DIABETES MELLITUS

Introduction

Diabetes Mellitus is a metabolic disorder characterized by hyperglycemia including polydipsia, polyphagia, polyuria and impaired vision due to defects in the insulin secretion and insulin action and both; occurs as a result of autoimmune, genetic factor, stress, sedentary lifestyle, destruction of insulin producing cells (β cells) in the pancreas. In 2020, International Diabetes Federation (IDF) estimated that in India 77 million people are suffering from diabetes that makes India the second most country affected with diabetes due to increased incidence of obesity, lifestyle changes, eating unhealthy food, low physical activity and consumption of alcohol. Diabetes Mellitus is classified in to two types namely type 1 and type 2 diabetes mellitus.

Type 1 diabetes mellitus, formerly known as juvenile diabetes or insulin-dependent diabetes usually occurs in childhood, teenagers and young adults before the age of 30 and it is an autoimmune disease caused by inadequate secretion of insulin due to destruction of pancreatic β cells possibly triggered by an environmental exposure in genetically susceptible individuals.

Type 2 diabetes mellitus formerly known as non-insulin dependent diabetes usually occurs in adults, but can occur even in children now a day due to childhood obesity. Usually it begins with insulin resistance which leads to elevated insulin secretion in order to maintain normal blood glucose levels. But in susceptible individuals, the pancreatic β cells are unable to sustain the increased demand for insulin and a slowly progressive insulin deficiency develops. Increasing age, obesity, lack of physical activity, hypertension, dyslipidemia and family history are the predisposing factors for type 2 diabetes.

Clinical features:

Initially, it may remain asymptomatic for a long period of time. On the other hand, in the later stage the features of long-term complications may manifest and become a part of the diabetic syndrome.

Type 1 diabetes mellitus is characterized by hyperglycemia associated with polyuria, polydipsia, polyphagia, weakness, weight loss, restlessness and it may lead to diabetic ketoacidosis with altered sensorium and severe dehydration.

Type 2 diabetes mellitus is characterized by hyperglycemia associated with polyuria, polydipsia, polyphagia, weakness, weight loss and it is often asymptomatic and remain silent for many years and at diagnosis may have features of long-term complications like neuropathy (tingling, numbness, paraesthesia of lower limbs), retinopathy and nephropathy. Sometimes, it may lead to hyperosmotic coma.

Diagnosis:

Diagnosis is based on oral glucose tolerance testing, fasting plasma glucose levels and glycosylated Hb (HbA1C).

The **Oral glucose tolerance test (OGTT)** is commonly employed for the diagnosis of prediabetes and diabetes, particularly in cases where gestational diabetes is a concern. Additionally, it is utilized to evaluate insulin resistance, impaired pancreatic beta cell function, and occasionally to identify reactive hypoglycemia, acromegaly, or other rare disorders of carbohydrate metabolism. The interpretation of results for type 2 diabetes screening is as follows:

- A 2-hour plasma glucose level <140 mg/dL is considered within normal limits
- A 2-hour plasma glucose level of 140-199 mg/dL signifies impaired glucose tolerance
- A 2-hour plasma glucose level ≥200 mg/dL indicates the presence of diabetes

To confirm a diagnosis, the test must be repeated on another day in close succession, yielding similar results. Alternatively, an alternative screening test may be employed for confirmation. A single anomalous OGTT result is inadequate for the diagnosis of diabetes or prediabetes.

The **Fasting plasma glucose test (FPG)**, or **Fasting Glucose test (FGT)**, is a widely used to screen for prediabetes, and diabetes. This test is conducted after fasting overnight, typically for 8-12 hours. The normal fasting blood glucose levels are

- 99 mg/dL or lower: Normal fasting blood sugar level
- 100–125 mg/dL: Higher than normal blood sugar levels, may indicate prediabetes
- 126 mg/dL or higher: High blood sugar level, may indicate diabetes

The **Glycosylated hemoglobin (HbA1c)** test, also known as the **Glycated hemoglobin** or **A1c test**, is used to assess a person's glucose control. This test provides an average of the blood sugar level over the past 90 days as a percentage. It can also be used to diagnose diabetes.

- A normal HbA1c value is below 5.7%.
- A value between 5.7% and 6.4% indicates prediabetes.
- Diabetes can be diagnosed with an HbA1c value of 6.5% or higher.

PANCREATITIS

Introduction

Pancreatitis is a condition characterised by the inflammation of the pancreas, an extensive gland located behind the stomach responsible for producing digestive enzymes and various hormones. The affliction is categorized into two primary types, namely, acute pancreatitis and chronic pancreatitis. Globally, the incidence rate of acute pancreatitis averages at approximately 34 cases per 100,000 individuals annually, while chronic pancreatitis exhibits a lower incidence ranging from 5 to 12 cases per 100,000 individuals per year. In India, the prevalence of chronic pancreatitis presents significant regional variance, with estimates ranging from 114 to 200 cases per 100,000 individuals, particularly prevalent in the southern states. The estimated incidence of acute pancreatitis in India ranges from 13 to 45 cases per 100,000 individuals annually, while chronic pancreatitis demonstrates a lower incidence ranging from 5 to 31.7 cases per 100,000 individuals per year.

Acute pancreatitis is commonly caused by gallstones and heavy alcohol use. Other causes include autoimmune pancreatitis, hypertriglyceridemia, post-endoscopic retrograde cholangiopancreatography (ERCP), hereditary factors, pancreatic duct injury, and certain medications (azathioprine, oral contraceptives, corticosteroids, etc.). Rare causes include biliary sludge and microlithiasis, biliary obstruction, hypercalcemia, infections like mumps, coxsackievirus, hepatitis B, etc., toxins, vascular diseases leading to pancreatic ischemia, anatomic abnormalities like choledochal cysts, and idiopathic causes.

Chronic pancreatitis is commonly caused by long-term alcohol abuse. Smoking is also a significant risk factor. Other causes include tropical pancreatitis, unknown causes, ductal blockage, genetic mutations related to cystic fibrosis, hereditary pancreatitis, and systemic diseases such as systemic lupus erythematosus.

The pathogenesis of acute pancreatitis can be attributed to several factors. These include damage to the pancreatic duct and acinar. In cases of acute pancreatitis, inadequate secretion of pancreatic digestive enzymes can lead to self-digestion and inflammation. Additionally, alcohol can trigger acute pancreatitis through direct toxicity and immunological processes.

Gallstones can lead to temporary blockage of the pancreatic duct, and this mechanism is also believed to be responsible for ERCP-induced pancreatitis. Furthermore, chronic pancreatitis can result from repeated acute attacks, leading to inflammatory infiltrates and fibrosis within the pancreas. Ultimately, this results in pancreatic insufficiency, which is irreversible and progressive.

Clinical features:

Acute pancreatitis commonly manifests with abdominal pain localized to the epigastric region that often radiates to the back. The associated discomfort is characteristically severe and sharp and frequently coincides with symptoms of nausea and vomiting.

Chronic pancreatitis may manifest with symptoms such as abdominal pain, nausea, and vomiting; however, it can also present as a painless condition. In such instances, patients may exhibit manifestations including steatorrhea and unintended weight loss.

Complication:

Complications of acute pancreatitis can manifest as peripancreatic fluid collection, pseudocyst or necrosis, cardiovascular failure, acute respiratory distress syndrome, acute kidney injury, shock, and disseminated intravascular coagulation. Meanwhile, complications of chronic pancreatitis include pseudocyst formation, diabetes, pseudoaneurysms, splenic vein thrombosis, and recurrent acute attacks.

PANCREATITIS-INDUCED DIABETES MELLITUS

Pancreatitis-induced Diabetes Mellitus, also referred to as Pancreatogenic diabetes mellitus (Type 3c Diabetes), is a form of secondary diabetes stemming from pancreas damage. This damage can be attributed to various conditions such as chronic pancreatitis, pancreatic surgery, cystic fibrosis, hemochromatosis, and pancreatic cancer.

Type 3c Diabetes mellitus is characterized by both exocrine and endocrine dysfunctions of the pancreas.

Exocrine Dysfunction: Chronic inflammation leads to the deterioration of acinar cells, resulting in exocrine pancreatic insufficiency, which adversely impacts nutrient digestion and absorption. Fibrosis (scarring) and calcification (stones) within the pancreas due to chronic pancreatitis further compromise pancreatic function (Fibrocalculous Pancreatic Diabetes).

Endocrine Dysfunction: Damage or depletion of the islets of Langerhans leads to decreased production and secretion of insulin and glucagon. Reduced insulin and glucagon levels contribute to inadequate regulation of blood glucose levels, leading to episodes of hyperglycemia and hypoglycemia.

Clinical Features:

The symptoms associated with Type 3c diabetes are akin to those present in other forms of diabetes. These include increased thirst (polydipsia) and dry mouth, frequent urination, fatigue, blurred vision, unexplained weight loss, as well as numbness or tingling in the extremities. Additionally, individuals with Type 3c diabetes commonly exhibit symptoms of exocrine pancreatic insufficiency, encompassing abdominal pain, gas and bloating, constipation, diarrhoea, steatorrhea (fatty stools), and malnutrition.

Diagnosis:

In Acute pancreatitis, diagnosis is based on clinical assessment, Serum amylase and lipase levels, and imaging studies.

Clinical examination reveals abdominal tenderness/guarding and decreased bowel sounds if an ileus is present. Grey-Turner sign (ecchymosis in the flanks) and Cullen's sign (ecchymosis around the umbilicus) are present.

Elevated serum amylase (normal level is 30-110U/L) and lipase (normal level is 0-160U/L) levels on the first day of acute pancreatitis, which generally normalize within 3 to 7 days, serve as key indicators. Lipase is particularly specific for pancreatitis. Imaging modalities such as CT with intravenous contrast play a pivotal role in the diagnosis of acute pancreatitis and the evaluation of local complications. Moreover, abdominal ultrasonography and chest x-rays may be utilized for diagnostic purposes.

The American College of Gastroenterology stipulates that for a diagnosis of acute pancreatitis, should include at least two of the following criteria is necessary:

- Abdominal pain consistent with acute pancreatitis,
- A serum lipase level at least three times the upper limit of the normal range, and
- Findings of acute pancreatitis on abdominal imaging

Conversely, in chronic pancreatitis, serum amylase and lipase levels are typically within normal limits and are of limited diagnostic value. Imaging modalities, such as CT abdomen, Magnetic resonance cholangiopancreatography (MRCP), and Endoscopic retrograde cholangiopancreatography (ERCP), are pivotal in diagnosis of chronic pancreatitis. Endoscopic ultrasonography is also efficacious in detecting subtle abnormalities in the pancreatic parenchyma and duct.

In Type 3c Diabetes mellitus, clinical assessment is pivotal in identifying the presence of chronic pancreatitis or prior pancreatic surgery, alongside physical examinations to discern signs of pancreatic insufficiency. Screening methods encompass the measurement of blood glucose and pancreatic enzyme levels, as well as the administration of an oral glucose tolerance test (OGTT). Additionally, imaging studies such as CT or MRI scans play a crucial role in detecting structural abnormalities such as pancreatic calcification and fibrosis, particularly in cases of Fibrocalculous Pancreatic Diabetes.

MCQ's

1. Which of the following is NOT a common symptom of Diabetes mellitus
   - a) Polydipsia
   - b) Polyphagia
   - c) Polyuria
   - d) Dysuria
2. What is the primary cause of Type 1 Diabetes Mellitus
   - a) Insulin resistance
   - b) Autoimmune destruction of pancreatic β cells
   - c) Obesity
   - d) Sedentary lifestyle
3. Which of the following is a predisposing factor for Type 2 Diabetes Mellitus
   - a) Childhood
   - b) Autoimmune disease
   - c) Increasing age
   - d) Genetic mutation
4. Which diagnostic test is commonly used to evaluate insulin resistance and impaired pancreatic beta cell function
   - a) Fasting plasma glucose levels
   - b) Glycosylated Hb
   - c) Oral glucose tolerance test
   - d) All of the above
5. According to the interpretation of the Oral Glucose Tolerance Test (OGTT), a 2-hour plasma glucose level of 140-199 mg/dL signifies
   - a) Normal glucose tolerance
   - b) Impaired glucose tolerance
   - c) Diabetes
   - d) Hypoglycemia
6. What is the normal fasting blood glucose level according to fasting plasma glucose test
   - a) 99mg/dL or lower
   - b) 100-125mg/dL
   - c) 126mg/dL or higher
   - d) 140mg/dL or higher
7. Diabetes can be diagnosed with an HbA1c value of
   - a) Below 5.7%
   - b) 5.7% to 6.4%
   - c) 6.5% or higher
   - d) 7.0% or higher
8. What is a significant risk factor for chronic pancreatitis
   - a) Gallstones
   - b) Smoking
   - c) Hypercalcemia
   - d) Infections

9. Which of the following is NOT a typical clinical finding in acute pancreatitis
   - a) Abdominal tenderness
   - b) Grey-Turner sign
   - c) Cullen's sign
   - d) Elevated Serum Creatinine
10. Which of the following is a common clinical feature of acute pancreatitis
    - a) Steatorrhea
    - b) Unintended weight loss
    - c) Epigastric pain radiating to the back
    - d) Painless abdomen
11. Which of the following conditions is NOT a cause of pancreatitis-induced diabetes mellitus
    - a) Chronic pancreatitis
    - b) Pancreatic surgery
    - c) Cystic fibrosis
    - d) Type 1 diabetes
12. What is the primary characteristic of exocrine dysfunction in Type 3c diabetes
    - a) Increased insulin production
    - b) Decreased glucagon production
    - c) Exocrine pancreatic insufficiency
    - d) Increased nutrient absorption
13. Which of the following is a symptom of exocrine pancreatic insufficiency in Type 3c diabetes
    - a) Polydipsia
    - b) Steatorrhea
    - c) Blurred vision
    - d) Frequent urination
14. What leads to the endocrine dysfunction in Type 3c diabetes
    - a) Increased insulin secretion
    - b) Damage or depletion of the islets of Langerhans
    - c) Increased glucagon secretion
    - d) Enhanced nutrient digestion
15. Which of the following is a common symptom of exocrine pancreatic insufficiency in individuals with Type 3c diabetes
    - a) Abdominal pain
    - b) Increased energy levels
    - c) Enhanced digestion
    - d) Improved nutrient absorption

Answer Keys

1	d	2	b	3	c	4	c	5	b	6	a	7	c	8	b
9	d	10	c	11	d	12	c	13	b	14	b	15	a		

Short Answer Questions

1. Explain Diabetes Mellitus in detail
2. Explain Pancreatitis in detail
3. Describe Pancreatogenic diabetes mellitus

TOPIC 40

Vātavyādhi

Introduction

Vāta is Āyu (longevity), Bala (strength), Vāyurdhāta śarīriṇaṃ (sustainer of the body of all living beings), Viśvata (all pervasive), Prabhutā (superior among all in the universe) and it is responsible for Sthita (existence), Utpatti (origin) and Vināśa (destruction) of all living creatures. Vāta is also responsible for all the activities of the body and even Pitta, Kapha, Sapta dhātus and Trimalas are also controlled by Vāta. Vāta in its own place with its normal functions is responsible for healthy life span of hundred years without any diseases in human being and also it causes immediate death if it becomes impaired and moves in abnormal path.

Vāta Vyādhi is a group of disorders caused by Vāta doṣa alone and can also be known as Vātaja Nānātmaja Vyādhi. Hence all the Ācāryas have given importance and dedicated independent chapters to it. Ācārya Caraka explained Vāta Vyadhi after Ūrustambha because Rūkṣaṇa karma (roughening therapy) in Ūrustambha vitiates the Vāta doṣa. Ācārya Vāgbhaṭa described Vāta Vyadhi after Kuṣtha because Vāta Vyādhi has Bahuprakāra (many classifications) like Kuṣṭha roga. Ācārya Suśruta described Vāta Vyādhi in two chapters namely Vātavyādhi Cikitsā and Mahāvāta vyādhi Cikitsā because of the severity and difficulty in treating these diseases.

Nirūkti and Paribhāṣā

- ⊙ वातेन जनितो व्याधिर्वातव्याधिः । (Ma.M.N.22/1)

The disease which is caused by vitiation of Vāta is called Vāta Vyādhi

- ⊙ वातजनितोऽसाधारणव्याधिर्वातव्याधिरिति । (Ma.M.N.22/1)

The specific or uncommon diseases caused by vitiation of Vāta is called Vāta Vyādhi

- ⊙ तत्र वात एव व्याधिर्वातव्याधिरिति पक्षे वात एव तत्तद्दूष्यसम्प्राप्तः सर्वाङ्गैकाङ्गरूपतामापन्नो विविधपीडाकर्तृत्वाद्वातव्याधिरुच्यते; यदा तु वाताद्व्याधिर्वातव्याधिरिति पक्षस्तदा वातदोषाद् दोषदूष्यसम्मूर्च्छनविशेषरूपः सर्वाङ्गैकाङ्गरूपो व्याधिर्वातव्याधिरुच्यते। (Ca.Ca.Ci.28/1)

Ācārya Cakrapāṇi interpreted the word Vātavyādhi in two ways

- ◈ 'Vāta eva vātavyādhi' - Vāta itself is a disease. Here it is implied that the aggravated Vāta itself after combining with the particular dūṣyas, produces Vividha pīḍa (different types of pain) over Sarvāṅga (whole body) or Ekāṅga (particular part of the body). Hence named Vātavyādhi.
- ◈ 'Vātadvyādhirvātavyādhi' - Disease caused by Vāta is Vātavyādhi. Here it is implied that Viśeṣarūpa doṣadūṣya sammūrcchana (doṣa and dūṣya interact in a special way) takes place due to Vāta which results in disease of Sarvāṅga (whole body) or Ekāṅga (particular part of the body). Hence named Vātavyādhi.

Sāmānya Nidāna and Saṃprāpti of Vāta Vyādhi

⊙ रूक्षशीताल्पलघ्वन्नव्यवायातिप्रजागरैः। विषमादुपचाराच्च दोषासृक्स्रवणादति॥
लङ्घनप्लवनात्यध्वव्यायामातिविचेष्टितैः। धातूनां सङ्क्षयाच्चिन्ताशोकरोगातिकर्षणात्॥
दुःखशय्यासनात् क्रोधाद्दिवास्वप्नाद्भयादपि। वेगसन्धारणादामादभिघातादभोजनात्॥
मर्माघाताद्गजोष्ट्राश्वशीघ्रयानापतंसनात्। देहे स्रोतांसि रिक्तानि पूरयित्वाऽनिलो बली॥
करोति विविधान् व्याधीन् सर्वाङ्गैकाङ्गसंश्रितान्। (Ca.Ci.28/15-19)

Intake of Rūkṣa (non-unctuous substances), Śīta (cold substances), Alpa (less in quantity), Laghvanna (light diet); indulging in Ativyavāya (excessive sexual intercourse), Prajāgaraiḥ (remaining awake at night time), Viṣamādupacāra (inappropriate application of therapeutic measures), Doṣāsṛksravaṇāt (excessive elimination of Doṣas including Malas and Rakta), Laṅghana (fasting), Plavana (swimming), Atyadhva (excessive walking), Ativyāyāma (excessive exercise), Aticeṣṭita (excessive physical exertion), Dhātunāṃ saṃkṣayāt (depletion of dhātus), Cintāśokarogātikarṣaṇāt (excessive emaciation due to worries, grief and diseases), Duḥkhaśayyāsanāt (sleeping or sitting over uncomfortable beds or seats), Krodhāt (anger), Divāsvapnā (sleeping during day time), Bhayāt (fear), Vegasaṃdhāraṇāt (suppression of natural urges), Āmāt (Āma doṣa), Abhighātāt (injury), Abhojanāt (abstinence from food), Marmāghātāt (injuries to vital parts of the body) and Gajoṣṭrāśvaśīghrayānāpataṃsanāt (riding over an elephant, camel, horse or fast moving vehicles and falling from the seat while riding on these animals and vehicles) leads to aggravation of Vāta, which fills up the Riktāni deha srotāṃsi (empty channels of the body) and manifests various diseases affecting the Sarvāṅga (entire body) or Ekāṅga (a specific part of the body).

Saṃprāpti Ghaṭaka of Sāmānya Vāta Vyādhi

Doṣa	Vāta
Dūṣya	Sapta dhātus, Malas

Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Agni māndhya janya Āma
Adhiṣṭhāna	Sarvāṅga and Ekāṅga
Srotas	Sarva Rikta srotas
Srotoduṣṭi	Saṅga, Atipravṛtti and Vimārga gamana
Udbhava sthāna	Pakvāśaya
Sañcāra sthāna	Sarva Rasāyanis
Vyakta sthāna	Sarvāṅga and Ekāṅga
Rogamārga	Madhyama

Sāmānya Pūrvarūpa of Vāta Vyādhi

- अव्यक्तं लक्षणं तेषां पूर्वरूपमिति स्मृतम्॥ आत्मरूपं तु तद्व्यक्तमपायो लघुता पुनः। (Ca.Ci.28/19-20)

Avyakta rūpa (indistinct manifestations of clinical features) of the diseases are known as premonitory symptoms. When these features are distinctly manifested, they are called Ātmarūpa (specific clinical feature of the disease). Laghutā i.e. lightness of these features indicates the alleviation of the disease.

Bheda of Vāta Vyādhi

- वायोर्धातुक्षयात् कोपो मार्गस्यावरणेन च (वा)। (Ca.Ci.28/59)

Vāta Vyādhi is broadly classified into two types based on Samprāpti (pathogenesis) namely Dhātukṣayaja or Nirupastambhita and Mārgāvaraṇaja or Upastambhita Vāta Vyādhi

- **Dhātukṣayaja Vāta Vyādhi** is further classified into two types based on Sthāna (location), namely Dhātugata and Anyasthānagata.
    - **Dhātugata Vāta Vyādhis** are Rasa (Tvak) gata, Raktagata, Māṃsagata, Medogata, Asthigata, Majjagata and Śukragata.
    - **Āśayagata or Anyagata Vāta Vyādhis** are Koṣṭhagata, Sarvāṅga gata, Gudagata, Āmāśaya gata, Pakvāśaya gata and Indriyagata
- **Mārgāvaraṇaja Vāta Vyādhi** is further classified into two types namely Anya Āvaraṇa and Anyonya or Paraspara Āvaraṇa
    - **Anya Āvaraṇa (Mūrta Āvaraṇa)** is obstruction of Vāta by other doṣas, dhātus and malas and it is of 22 types, they are Pittāvṛta, Kaphāvṛta and Pittakaphāvṛta; Doṣāvṛta Pañcavāta (Pittāvṛta Pañcavāta - 5, Kaphāvṛta Pañcavāta - 5); Dhatvāvṛta Vāta (Raktāvṛta, Māmsāvṛta, Medāvṛta, Asthyāvṛta, Majjāvṛta and Śukrāvṛta); Annāvṛta Vāta, Malāvṛta Vāta and Mūtrāvṛta Vāta..

Ācārya Vāgbhaṭa added Āmāvṛta Vāta and Sarvadhātvāvṛta Vāta, whereas Ācārya Suśruta described ten types of Āvaraṇa i.e. Pittāvṛta Pañcavāta and Kaphāvṛta Pañcavāta.

- **Anyonya or Paraspara Āvaraṇa (Amūrta Āvaraṇa)** is obstruction of Vāta by other Vāta, and it is of 20 types, they are Prāṇāvṛta udāna, Prāṇāvṛta samāna, Prāṇāvṛta vyāna, Prāṇāvṛta apāna, Udānāvṛta prāṇa, Udānāvṛta samāna, Udānāvṛta vyāna, Udānāvṛta apāna, Samānāvṛta prāṇa, Samānāvṛta udāna, Samānāvṛta vyāna, Samānāvṛta apāna, Vyānāvṛtaprāṇa, Vyanāvṛta udāna, Vyanāvṛta samāna, Vyanāvṛta apāna, Apānāvṛta prāṇa, Apānāvṛta udāna, Apānāvṛta samāna and Apānāvṛta vyāna.

Vāta Vyādhi is again classified into two types based on Saṃsthāna (region) i.e. Sarvāṅga (entire body) or Ekāṅga (a specific part of the body).

- **Sarvāṅga Vāta Vyādhis** are Ākṣepaka, Apatantraka, Apatānaka, Daṇḍāpatānaka, Dhanuḥstambha, Ābhyantarāyāma, Bahyāyāma, Pakṣāvadha, Sarvāṅgavadha and etc.
- **Ekāṅga Vāta Vyādhis** are Ardita, Hanugraha, Manyastambha, Jihvāstambha, Gṛdhrasī, Viṣvācī, Kroṣṭukaśīrṣa, Vātakaṇṭaka, Pādadāha, Pādaharṣa and etc.

Sāmānya Lakṣaṇās of Vāta Vyādhi

- सङ्कोचः पर्वणां स्तम्भो भेदोऽस्थ्नां पर्वणामपि॥ लोमहर्षः प्रलापश्च पाणिपृष्ठशिरोग्रहः।
खाञ्ज्यपाङ्गुल्यकुब्जत्वं शोषोऽङ्गानामनिद्रता॥ गर्भशुक्ररजोनाशः स्पन्दनं गात्रसुप्तता।
शिरोनासाक्षिजत्रूणां ग्रीवायाश्चापि हुण्डनम्॥ भेदस्तोदार्तिराक्षेपो मोहश्चायास एव च।
एवंविधानि रूपाणि करोति कुपितोऽनिलः॥ हेतुस्थानविशेषाच्च भवेद्रोगविशेषकृत्। (Ca.Ci.28/20-24)

Saṅkoca (contracture), Parvāṇāṃ stambhā (stiffness of small joints), Bhedoasthnāṃ parvāṇam (tearing type of pain in bones and joints), Lomaharṣa (horripilation), Pralāpa (delirious speech), Pāṇipṛṣṭhaśirograha (stiffness of hand, back and head), Khañja (limping), Paṅgu (lameness), Kūbjatva (humpbacked), Aṅgaśoṣa (wasting of body parts), Anidratā (loss of sleep), Garbhaśukrarajonāśa (destruction of embryo, semen and menses), Spandana (pulsation), Gātra suptatā (numbness in the body), Śironāsākṣijatrūṇāṃ grīvāyāścāpi huṇḍana (paralyzed in head, nose, eyes, clavicular region and neck), Bheda (tearing pain), Toda (pricking pain), Arti (excruciating pain), Ākṣepa (convulsions), Moha (unconsciousness) and Āyāsa (fatigue) are the general clinical features manifested by aggravated Vāta. The specific features are produced by aggravated Vāta due to specificity of cause and location.

Sādhyāsādhyata

- ⊙ सन्धिच्युतिर्हनुस्तम्भः कुञ्चनं कुब्जताऽर्दितः॥ पक्षाघातोऽङ्गसंशोषः पङ्गुत्वं खुडवातता।
स्तम्भनं चाढ्यवातश्च रोगा मज्जास्थिगाश्च ये॥ एते स्थानस्य गाम्भीर्याद्यत्नात् सिध्यन्ति वा न वा।
नवान् बलवतस्त्वेतान् साधयेन्निरुपद्रवान्॥ (Ca.Ci.28/72-74)

Sandhicyuti (dislocation of joints), Hanustambha (lock jaw), Kuñjana (contracture), Kubjatā (hunch back), Ardita (facial paralysis), Pakṣāghāta (hemiplegia), Aṅgasaṃśoṣa (wasting of body part), Paṅgutva (lameness), Khuḍavāta (Gulphavāta – Vāta disorder of ankle joint), Stambhana (stiffness), Āḍhyavāta (Medavṛta Vāta) and Majjāsthyāvṛta Vāta, all these diseases are usually difficult to cure because of their deep seated nature as they get firmly located in these seats of manifestations. Though, it may be cured if it is Nava (recently manifested) in the Balavān (strong person) with Nirupadrava (absence of complications).

Short Answer Questions

1. Explain Sāmānya Nidāna and Saṃprāpti of Vātavyādhi
2. Explain Sāmānya Lakṣaṇās of Vātavyādhi

Long Answer Questions

1. Explain the Sāmānya Nidāna, Saṃprāpti, Pūrvarūpa and Lakṣaṇās of Vātavyādhi.

◆

TOPIC 41

Snāyugata Vāta

Introduction

The Snāyu, regarded as a ligament, serves as a binding structure within the body. It originates from the Snigdha bhāva of Medas (fat) and binds the Māṃsa (muscles), Asthi (bones), and Meda (fat) with substantial potency. These bindings are stronger than Sirā (blood vessels) and can bind the joints very strongly. There are nine hundred Snāyus in total, with six hundred in the Śākhā (extremities), two hundred and thirty in the Koṣṭha, and seventy in the Grīvā (neck) and above. Snāyu is classified into four types: Pratāna (branched), Vṛtta (round), Pṛthu (flat), and Śuṣira (porous). Ācārya Suśruta described the importance of Snāyu using a simile, comparing it to a boat built with tightly fastened wooden planks that can carry weight in water. Similarly, the human body can carry weight as long as the joints are tightly fastened by Snāyu in many ways. According to Suśruta, injuries to the Asthi (bones), Peśi (muscles), Sirā (blood vessels), and Sandhi (joints) do not cause as much disability as injuries to the Snāyu (ligaments).

- ⊙ बाह्याभ्यन्तरमायामं खल्लिं कुब्जत्वमेव च। सर्वाङ्गैकाङ्गरोगांश्च कुर्यात् स्नायुगतोऽनिलः॥ (Ca.Ci.28/35)
- ⊙ .....स्नावस्थितः कुर्याद्गृध्रस्यायामकुब्जताः। (A.H.Ni.15/14)
- ⊙ स्नायुप्राप्तः स्तम्भकम्पौ शूलमाक्षेपणं तथा ॥ (Su.Ni.1/27)

WhenvitiatedVātaislocatedintheSnāyu(ligaments),itproducesBāhyābhyantaramāyāmaṃ (ophisthotonos and emprosthotonos), Khalli (shooting pain in the extremities), Kubjatva (hunch backed), Gṛdhrasī (lumbago) and Sarvāṅgaikāṅga roga (Vātaja disorders involving whole body and one part of the body). Ācārya Suśruta added some other features like Stambha (stiffness), Kampa (tremors), Śūla (pain) and Ākṣepaṇa (convulsions).

Snāyugata Vātavyādhis are

- Āyāma or Bāhyābhyantaramāyāmaṃ
- Khalli,
- Kubjatva
- Gṛdhrasī
- Viśvācī
- Sarvāṅga roga such as Apatānaka, Ākṣepaka, Kampavāta
- Ekāṅga roga such as Pakṣāghāta, Ardita, Paṅgutva

ĀKṢEPAKA

Nirūkti and Paribhāṣa

The term Ākṣepaka is derived from the Sanskrit root 'Ā+kṣip' (आ+क्षिप्), which means "to throw" or "to hurl." The suffix 'ṇvul' (ण्वुल्) is added to form the noun, resulting in Ākṣepaka, which means one who throws, a detractor or a convulsant.

- मुहुरेव देहमाक्षिपति स आक्षेपणादाक्षेपक इति निरुक्तिः। (Ga.Su.Ni.50-51)

The individual who Muhureva dehamākṣipati (constantly or repeatedly throws or hurls the body) is referred to as Ākṣepaka, due to his involvement in Ākṣepaṇāt (the act of throwing or tossing).

- मुहुर्मुहुस्तदाक्षेपादाक्षेपक इति स्मृतः ॥ (Su.Ni.1/50-51)

Muhurmuhustadākṣepāt (repeated convulsions) is known as Ākṣepaka

- आक्षेपणाच्चालनादाक्षेपः, गात्रचालन इत्यर्थः । स च हस्त्यारूढस्येव गात्रं चालयतीत्यभिप्रायः । (Āḍ.Śā.P.7/105-114)
- आक्षिपति देहं हस्त्यारूढपुरुषस्येव गात्रं चालयति। (Ma.M.N.22/27)
- मुहुर्मुहुर्देहमाक्षिपति, हस्त्यारूढस्येव गात्रं चालयति। (Āt.M.N.22/27)

The term Ākṣepa is derived from Ākṣepaṇa, which means movement or throwing. It specifically connotes "Gātracālana" (movement of the body), suggesting that it simulates the physical movement of the body mounted on the elephant. In short, Ākṣepaka represents a vigorous and consequential movement similar to the body mounted on an elephant.

Ākṣepaka Saṃprāpti

- मुहुराक्षिपति क्रुद्धो गात्राण्याक्षेपकोऽनिलः॥ पाणिपादं च संशोष्य सिराः सस्नायुकण्डराः। (Ca.Ci.28/50-51)

The aggravated Vāta does Samśoṣaṇa (dries up) the Śirasnāyukaṇḍarāḥ (vessels, tendons and ligaments) of the Pāni (hands) and Pāda (legs) and causes repeated convulsions in different parts of the body; this is called Ākṣepaka.

- यदा तु धमनीः सर्वाः कुपितोऽभ्येति मारुतः ॥ तदाक्षिपत्याशु मुहुर्मुहुर्देहं मुहुश्चरः ।
  मुहुर्मुहुस्तदाक्षेपादाक्षेपक इति स्मृतः ॥ (Su.Ni.1/50-51)

The aggravated Vāta invades Sarvā dhamanīḥ (all the blood vessels) and produces Muhurmuhur Akṣiptyāśu (repeated convulsions quickly) throughout the body; because of Muhurmuhustadākṣepāt (repeated convulsions), it is known as Ākṣepaka.

Ākṣepaka can be predominantly characterised by conditions like convulsions.

Ākṣepaka Bheda

- कफपित्तान्वितो वायुर्वायुरेव च केवलः । कुर्यादाक्षेपकं त्वन्यं चतुर्थमभिघातजम् ॥ (Su.Ni.1/58)

Ākṣepaka is classified into four types namely Kevala Vātaja, Vāta pittaja, Vāta Kaphaja and Abhighātaja.

- आक्षेपकश्चतुर्धा- अपतानकः, संसृष्टाक्षेपकः, केवलाक्षेपकः, अभिघातजश्च; (Ḍa.Su.Ni.1/58)

Ācārya Ḍalhaṇa classified Ākṣepaka in to four types namely Apatānaka, Saṃsṛṣṭākṣepaka, Kevalākṣepaka and Abhighātaja.

- चरकेण आक्षेपक एव दण्डापतानकोऽभ्यन्तरायामो बहिरायाम आगन्तुक इति चतुर्विध उक्तस्तेनाभिघातेन व्रणकरत्वादत्र व्रणायामः कथितः । (Āḍ.Śā.P.7/105-114)
- आक्षेपकश्चतुर्विधो भवति– दण्डापतानकोऽभ्यन्तरायामो बहिरायामोऽभिघातजश्चेति। (Ma.M.N.22/28-31)

Ācārya Āḍhamalla described that Ācārya Caraka classified Ākṣepaka into four types, namely, Daṇḍāpatānaka, Abhyantarāyāma, Bahirāyāma and Āgantuka or Vraṇāyāma.

Apatānaka or Apatantraka

- तत्रापतानकः 'सोऽपतानक' इत्यादिना 'वक्षःकट्यूरुभञ्जनम्' इति पर्यन्तेनोक्तः, । (Ḍa.Su.Ni.1/58)

Apatānaka is defined as Vakṣaḥkaṭyūrubhañjanam (breaking or injuring of the Chest, waist and thighs).

- आक्षेपस्यावस्थाविशेषो ऽपतानको भवति । (Āḍ.Śā.P.7/105-114)

Apatānaka is Avasthā viśeṣa (specific condition) of Ākṣepaka

- सोऽपतानकसञ्ज्ञो यः पातयत्यन्तराऽन्तरा । (Su.Ni.1/52)

◉ सोऽपतानक इत्यादि। य आक्षेपकोऽन्तराऽन्तरा पातयति सोऽपतानक इत्यर्थः। (Ḍa.Su.Ni.1/52)

Apatānaka is a special kind of Ākṣepaka, in which the person falls on the ground repeatedly with convulsions.

Apatānaka and Apatantraka Lakṣaṇās

◉ दृष्टिं संस्तभ्यं संज्ञाश्च हत्वा कण्ठेन कूजाति । हृदि भुक्ते नरः स्वास्थ्यं याति मोहं वृते पुनः ।
वायुना दारुणं प्राहुरेके तमपतानकम् ॥ (B.P.M.24/198)

In Apatānaka, Vāta gets aggravated and produces Dṛṣṭi saṃstabhya (fixed gaze), Saṃjña hatvā (loss of consciousness) and Kaṇṭhena kūjana (cooing sound from the throat); the person becomes Svāsthya (feels healthy) while taking Hṛdi bhojana (pleasing food to heart) and enters again in to state of Moha (loss of consciousness) while stopping the food.

◉ अधः प्रतिहतो वायुर्व्रजन्नूर्ध्वं हृदाश्रिताः॥ नाडीः प्रविश्य हृदयं शिरः शङ्खौ च पीडयन्।
आक्षिपेत्परितो गात्रं धनुर्वच्चास्य नामयेत्॥ कृच्छ्रादुच्छ्वसिति स्तब्धस्रस्तमीलितदृक्ततः।
कपोत इव कूजेच्च निःसंज्ञः सोऽपतन्त्रकः॥ स एव चापतानाख्यो मुक्ते तु मरुता हृदि।
अश्नुवीत मुहुः स्वास्थ्यं मुहुरस्वास्थ्यमावृते। (A.H.NI.15/17-20)

◉ वायुरूर्ध्वं व्रजेत् स्थानात् कुपितो हृदयं शिरः । शङ्खौ च पीडयत्यङ्गान्याक्षिपेन्नमयेच्च सः ॥
निमीलिताक्षो निश्चेष्टः स्तब्धाक्षो वाऽपि कूजति । निरुच्छ्वासोऽथवा कृच्छ्रादुच्छ्वस्यान्नष्टचेतनः ॥
स्वस्थः स्याद्धृदये मुक्ते ह्यावृते तु प्रमुह्यति । कफान्वितेन वातेन ज्ञेय एषोऽपतन्त्रकः ॥ (Su.Ni.1/64-66)

In Apatantraka, the aggravated Vāta gets obstructed in its downward movement, starts moving upwards and enters the vessels of the heart and afflicts the Hṛdaya (heart), Śiras (head), Śaṅkha (temporal region) and produces Ākṣipetparito gātraṃ (convulsions throughout the body) and Dhanurvaccāsya nāmayet (makes the body to bend like a bow), where the patients suffers from Kṛcchrāducchvāsiti (difficulty in breathing), Stabdhasrastamīlitadṛktataḥ (his eyes become fixed, drooping down and closed) and Kapota iva kūjana (makes sounds like pigeon), Niḥsaṃjña (becomes unconscious). This disease is also known as Apatānaka, in which the person become Svāsthya (feels healthy) if the heart becomes free from Vāta temporarily and become Asvāsthya (unhealthy) if the heart becomes afflicted or covered with Vāta.

Ācārya Suśruta mentioned that when the Kapha gets mixed up with Vāta, it is called Apatantraka.

Apatānaka Sādhyāsādhyata

◉ गर्भपातसमुत्पन्नः शोणितातिस्रवोत्थितः। अभिघातसमुत्थश्च दुश्चिकित्स्यतमो हि सः॥ (A.H.NI.15/21)

◉ गर्भपातनिमित्तश्च शोणितातिस्रवाच्च यः । अभिघातनिमित्तश्च न सिध्यत्यपतानकः ॥ (Su.Ni.1/59)

If Apatānaka occurs due to Garbhapāta (abortion), Śoṇitātisravā (excessive bleeding) and Abhighātaja (trauma), then it becomes Asādhyata (incurable).

Apatānaka or Apatantraka can be predominantly characterised by conditions like tetanic convulsions, hysteria, etc. **Yoṣāpatantraka** is a condition explained in Mādhava nidāna pariśiṣṭa, Śāraṅgadhara saṃhitā pariśiṣṭa, Bhaiṣajya Ratnāvalī etc., commonly seen in females. It is also known as Yoṣāpasmāra and it can be correlated with hysterical neurosis.

Daṇḍāpatānaka or Daṇḍaka

◉ कफान्वितो भृशं वायुस्तास्वेव यदि तिष्ठति ॥ स दण्डवत् स्तम्भयति कृच्छ्रो दण्डापतानकः । (Su.Ni.1/52-53)

◉ कफावृतो यदा वायुर्धमनीष्वेव तिष्ठति । स दण्डवत्स्तम्भयति कृच्छ्रो दण्डापतानकः ॥ (B.P.M.24/170)

In Daṇḍāpatānaka, Kapha covers the Vāta or the aggravated Vāta associated with Kapha afflicts the Dhamanī (blood vessels) and produces Daṇḍavat stambha (stiff like rod) and it is Kṛcchra Sādhya (difficult to cure).

◉ आमबद्धायनः कुर्यात्संस्तभ्याङ्गं कफान्वितः। असाध्यं हतसर्वेहं दण्डवद्दण्डकं मरुत्॥ (A.H.Ni.15/42)

When Vata doṣa is obstructed by Āma (toxins) and combined with Kapha, it causes Stabhyāṅga (stiffness in the body). This condition is considered Asādhya (incurable) and affects the entire body, making it Daṇdavaddaṇḍaka (hard and immobile like a stick).

◉ पाणिपादशिरःपृष्ठश्रोणीः स्तभ्नाति मारुतः॥ दण्डवत्स्तब्धगात्रस्य दण्डकः सोऽनुपक्रमः। (Ca.Ci.28/51-52)

In Daṇḍaka, the aggravated Vāta produces Pāṇipādaśiraḥpṛṣṭhaśroṇī stabhna (rigidity or stiffness of hands, legs, head, back and hips) and the body becomes Daṇḍavatstabdha (stiff like rod) and it is Anupakrama (untreatable).

Daṇḍāpatānaka and Daṇḍaka can be predominantly characterised by conditions like tonic clonic convulsions, tonic convulsion, and stiff person syndrome.

Dhanuḥstambha, Antarāyāma and Bahirāyāma

◉ धनुस्तुल्यं नमेद्यस्तु स धनुःस्तम्भसञ्ज्ञकः । अङ्गुलीगुल्फजठरहृद्वक्षोगलसंश्रितः ॥
स्नायुप्रतानमनिलो यदाऽऽक्षिपति वेगवान् । विष्टब्धाक्षः स्तब्धहनुर्भग्नपार्श्वः कफं वमन् ॥
अभ्यन्तरं धनुरिव यदा नमति मानवः । तदाऽस्याभ्यन्तरायामं कुरुते मारुतो बली ॥
बाह्यस्नायुप्रतानस्थो बाह्यायामं करोति च । (Su.Ni.1/54-57)

In Dhanuḥstambha, the aggravated Vāta bends the body like a bow and it is of two types namely Ābhyantarāyāma and Bāhyāyāma.

The aggravated Vāta gets lodged in the Snāyu of Aṅgulī (Pādagata - toes), Gulpha (ankle), Jaṭhara (abdomen), Hṛdaya (heart), Vakṣa (chest) and Gala (neck) resulting in sudden convulsive attack with Viṣṭabdhākṣa (fixed gaze), Stabdhahanu (stiffness of jaw), Bhagnapārśva (fractures in the flanks), Kaphavamana (vomiting of mucus) along with Abhyantara dhanuriva namati (bending inwardly like a bow); it is called Ābhyantarāyāma Dhanuḥstambha. On contrary, the aggravated Vāta gets lodged in the Bāhya Snāyu of Aṅgulī (Pādagata - toes), Gulpha (ankle), Jaṭhara (abdomen), Hṛdaya (heart), Vakṣa (chest) and Gala (neck) resulting in Bāhyāyāma Dhanuḥstambha (bending outwardly like a bow).

⦿ तमसाध्यं बुधाः प्राहुर्वक्षःकट्यूरुभञ्जनम् ॥ (Su.Ni.1/57)

If the patient of Dhanuḥstambha suffers from Vakṣaḥkaṭyūru bhañjana (fractures of chest, back and thigh), then it is Asādhya (incurable).

Ācārya Caraka described Antarāyāma as a synonym of Manyāstambha and Bahirāyāma as a synonym of Dhanustambha (Dhanuṣkambha).

⦿ मन्ये संश्रित्य वातोऽन्तर्यदा नाडीः प्रपद्यते। मन्यास्तम्भं तदा कुर्यादन्तरायामसञ्ज्ञितम्॥
अन्तरायम्यते ग्रीवा मन्या च स्तभ्यते भृशम्। दन्तानां दंशनं लाला पृष्ठायामः शिरोग्रहः॥
जृम्भा वदनसङ्गश्चाप्यन्तरायामलक्षणम्। (इत्युक्तस्त्वन्तरायामो बहिरायाम उच्यते)॥
पृष्ठमन्याश्रिता बाह्याः शोषयित्वा सिरा बली। वायुः कुर्याद्धनुस्तम्भं बहिरायामसञ्ज्ञकम्।
चापवन्नाम्यमानस्य पृष्ठतो नीयते शिरः। उर उत्क्षिप्यते मन्या स्तब्धा ग्रीवाऽवमृद्यते॥
दन्तानां दशनं जृम्भा लालास्रावश्च वाग्ग्रहः। जातवेगो निहन्त्येष वैकल्यं वा प्रयच्छति॥ (Ca.Ci.28/43-48)

The aggravated Vāta gets lodged in the Manya (nape of the neck) and reaches the Antaryadā nāḍī (internal nerves) and produces the disease Manyāstambha, which is also known as Antarāyāma. The characteristic features of Antarāyāma are Antarāyamyate grivā (inward bending of the neck) associated with Manyā stabhyate (stiffened nape of the neck), Dantānaṃ daṃśanaṃ (clinching of the teeth), Lālā sravā (salivation), Pṛṣṭhāyāma (back bends outwardly), Śirograha (stiffness of head), Jṛṃbhā (yawning) and Vadanasaṅga (inflexibility of the face).

The aggravated Vāta gets lodged in the Pṛṣṭhamanyā (back and nape of the neck) and leads **to Bāhya sirā śoṣa (drying up the external vessels) and produces the disease Dhanustambha, which is also known as Bahirāyāma. The characteristic features of Bahirāyāma are** Amyate pṛṣṭhato nīyate śiraḥ (the head bends backwards) associated with Ura utkṣipyate (lifting up of the chest), Manyā stabdhā (stiffened nape of the neck), Grīvāavamṛdyate (neck gets squeezed), Dantānaṃ daṃśanaṃ (clinching of the teeth), Jṛṃbhā (yawning), Lālā sravā (salivation) and Vāggraha (absence of speech). When it afflicts with force, it either kills or causes serious deformity.

⦿ विवर्णदन्तवदनः स्रस्ताङ्गो नष्टचेतनः। प्रस्विद्यंश्च धनुषम्भी दशरात्रं न जीवति॥
वेगेष्वतोऽन्यथा जीवेन्मन्देषु विनतो जडः। ख़ञ्जः कुणिः पक्षहतः पङ्गुलो विकलोऽथवा॥ (A.H.Ci.21/39-40)

If the patient of Dhanuṣkambha, suffers from Vivarṇa danta vadana (discoloration of teeth and face), Srastāṅga (body parts dropping down), Naṣṭacetana (loss of conciousness) and Prasvidya (excessive perspiration), then he will die within ten days. Contrary with above symptoms with mild paroxysm of Akṣepaka, he will survive with the complications such as Vinatā (bowed), Jaḍa (stiffness of the body), Khañja (loss of movement in one leg), Kuṇi (crooked arm), Pakṣāhata (loss of functions of half of the body) and Paṅgu (loss of movement in both legs) or Vikala (defective body parts).

Dhanuḥstambha can be predominantly characterised by conditions like Emprosthotonus (Antarāyāma - tetanic contraction of the flexor muscles, curving the back with concavity forward) and Opisthotonus (Bahirāyāma - tetanic spasm in which the spine and extremities are bent with convexity forward, the body resting on the head and the heels)

Vraṇāyāma

⦿ व्रणं मर्माश्रितं प्राप्य समीरणसमीरणात्। व्यायच्छन्ति तनुं दोषाः सर्वामापादमस्तकम्॥
तृष्यतः पाण्डुगात्रस्य व्रणायामः स वर्जितः। (A.H.Ni.15/27-28)

The aggravated doṣas gets lodged in the Marmāśrita Vraṇa (wound situated on the vital spots) due to aggravation of Vāta and it spreads all over the body and produces the disease Vraṇāyāma and it becomes incurable if it is associated with Tṛṣṇā (thirst) and Pāṇḍu gātra (yellowish white discoloration of the body).

⦿ गते वेगे भवेत्स्वास्थ्यं सर्वेष्वाक्षेपकेषु च॥ (A.H.Ni.15/28)

In all types of Ākṣepaka, the person recovers temporarily after occurrence of the episodes.

ARDITA OR EKĀYĀMA

Nirūkti and Paribhāṣa

⦿ मुखस्यार्धवक्रत्वेनोपलक्षितोऽर्दित इति प्रसिद्धः । (Āḍ.Śā.P.7/105-114)

That which is characterised by Mukhasyārdhavakratva (distortion or curvature in half of the face) is called Ardita.

⦿ अर्दयत्यनिलोवक्रमर्दितं जनयत्यतः ॥ (Āḍ.Śā.P.7/105-114)

That which is characterised by Vakramarditaṃ (distortion or curvature of the face) and Ardayati (severe pain) due to vitiated Vāta is called Ardita.

⦿ अर्धे तस्मिन्मुखार्धे वा केवले स्यात्तदर्दितम् । (Ga.Su.Ni.1/68-72)

That which occurs Ardhe (in one half), or Mukhārdhe (only in one half of the face), is called Ardita

⦿ अर्दितः-एकायामः। (He.A.H.Sū.2/18)

Ardita (distortion of one side of the face) is also known as Ekāyāmaḥ (indicating it affects only one side or one half of the face).

Nidāna and Saṃprāpti of Ardita

⦿ गर्भिणीसूतिकाबालवृद्धक्षीणेष्वसृक्क्षये । उच्चैर्व्याहरतोऽत्यर्थं खादतः कठिनानि वा ।
हसतो जृम्भतो भाराद्विषमाच्छयनादपि ॥ शिरोनासौष्ठचिबुकललाटेक्षणसन्धिगः ।
अर्दयित्वाऽनिलो वक्त्रमर्दितं जनयत्यतः ॥ (Su.Ni.1/68-69)

Due to indulging in Uccairvyāharatoatyartha (excessive shouting in loud voice), Khādataḥ kaṭhināni (chewing hard substances), Hasato (laughing), Jṛmbhato (yawning), Bhārāt (carrying heavy loads) and Viṣamācchayanāt (sleeping on uneven posture) by the Garbhiṇī (pregnant women), Sūtikā (women in puerperium), Bāla (children), Vṛddha (old aged), Kṣīṇa (emaciated) and Asṛk Kṣaya (who have lost blood), the Vāta aggravates extremely and gets lodged in the Śiras (head), Nāsa (nose), Oṣṭha (lips), Cibuka (chin), Lalāṭa (forehead) and Akṣaṇa sandhigaḥ (joints of the eye) and afflicts the Ardha Vaktra (half face) and produces the disease Ardita.

◉ अतिवृद्धः शरीरार्धमेकं वायुः प्रपद्यते। यदा तदोपशोष्यासृग्बाहुं पादं च जानु च॥
तस्मिन् सङ्कोचयत्यर्धे मुखं जिह्मं करोति च। वक्रीकरोति नासाभ्रूललाटाक्षिहनूस्तथा॥ (Ca.Ci.28/38-39)

The excessively aggravated Vāta afflicts Śarīrārdhamekaṃ (either right or left half of the body), then it does Upaśoṣana (dries up) of Asṛk (blood), Bāhu (hands), Pāda (legs) and Jānu (knee) and produces Atyarthā saṅkoca (severe contracture) resulting in Ardha mukha jihma (tortuousness of one side of face) and leads to Vakri nāsābhrūlalaṭākṣihanū (crooked nose, eyebrow, forehead, eye and jaw).

Pūrvarūpa of Ardita

◉ यस्याग्रजो रोमहर्षो वेपथुर्नेत्रमाविलम् ॥ वायुरूर्ध्वं त्वचि स्वापस्तोदो मन्याहनुग्रहः ।
तमर्दितमिति प्राहुर्व्याधिं व्याधिविशारदाः ॥ (Su.Ni.1/71-72)

Romaharṣa (horripilation), Vepathu (tremors), Netramāvilam (turbid eyes), Ūrdhva Vāyu (upward movement of Vāta), Tvaci svāpa (numbness of the skin), Toda Tvaci (pricking pain in the skin) and Manyāhanugraha (stiffened nape of the neck and jaw) are the premonitory symptoms of the disease Ardita by the physicians.

Sāmānya Lakṣaṇās of Ardita

◉ ततो वक्रं व्रजत्यास्ये भोजनं वक्रनासिकम् । स्तब्धं नेत्रं कथयतः क्षवथुश्च निगृह्यते॥
दीना जिह्मा समुत्क्षिप्ता कला सज्जति चास्य वाक्। दन्ताश्चलन्ति बाध्येते श्रवणौ भिद्यते स्वरः॥
पादहस्ताक्षिजङ्घोरुशङ्खश्रवणगण्डरुक् । अर्धे तस्मिन्मुखार्धे वा केवले स्यात्तदर्दितम्॥ (Ca.Ci.28/40-42)

Due to these, Vakraṃ vrajatyāsye bhojanaṃ (ingested morsel of food moves tortuously to one side of the mouth), Vakranāsikam stabdha netraṃ kathayataḥ (while expressing the nose become curved and eyes becomes fixed) and Kṣavathuśca nigṛhyate (sneezing urge gets suppressed). Dīnā jihmā samukṣiptā kalā sajjati cāsya vāk (speech becomes feeble, distorted, staggering, weak and imperceptible), Dantāścalanti (teeth become loose), Bādhyete śravaṇau (impaired hearing), Bhidyate svaraḥ (hoarseness of voice) and Pādahastākṣijaṅgoru śaṅkhaśravaṇagaṇḍaruk (pain in the leg, hand, eye, calf, thigh, temple, ear and cheek) and this condition may appears in Ardhe tasmin (half of the body) or Mukhārdhe (half of the face), and is called Ardita.

◉ वक्रीभवति वक्त्रार्धं ग्रीवा चाप्यपवर्तते । शिरश्चलति वाक्सङ्गो नेत्रादीनां च वैकृतम् ॥
ग्रीवाचिबुकदन्तानां तस्मिन् पार्श्वे तु वेदना । (Su.Ni.1/70-71)

Vakrībhavati vaktrārdhaṃ grīvā cāpyapavartate (half of the face and neck becomes crooked or distorted), Śiracalāti (shaking of the head), Vāksaṅga (absence of speech), Netrādīnāṃ vaikṛta (disfigurement of the eyes) and Grīvācibukadantānāṃ tasmin pārśva tu vedanā (pain in the neck, chin and teeth on the affected side).

◉ वक्रीकरोति वक्त्रार्धमुक्तं हसितमीक्षितम्। ततोऽस्य कम्पते मूर्द्धा वाक्सङ्गः स्तब्धनेत्रता॥
दन्तचालः स्वरभ्रंशः श्रुतिहानिः क्षवग्रहः। गन्धाज्ञानं स्मृतेर्मोहस्त्रासः सुप्तस्य जायते॥
निष्ठीवः पार्श्वतो यायादेकस्याक्ष्णो निमीलनम्। जत्रोरूर्ध्वं रुजा तीव्रा शरीरार्धेऽधरेऽपि वा॥
तमाहुरर्दितं केचिदेकायाममथापरे। (A.H.Ni.15/34-37)

Vāgbhaṭa added some other features of Ardita or Ekāyāma such as Danta cāla (shaking of the teeth), Svarabhraṃśa (hoarseness of voice), Śrutihāni (deafness), Kṣavagraha (obstruction of sneezing), Gandhājñānaṃ (loss of smell sensation), Smṛte (loss of memory), Moha (delusion), Trāsa suptasya (frightened during sleeping), Niṣṭhivaḥ pārśvato (spit comes out from the sides of the mouth), Ekasyākṣaṇo nimīlanam (closing of one eye) and Jatrūrdhva ruja tīvrā śarīrārdhereapivā (severe pain in the upper parts of the body or half of the body or lower parts of the body).

◉ स्वस्थः स्यादर्दितादीनां मुहुर्वेगे गतेऽगते॥ पीड्यते पीडनैस्तैस्तैर्भिषगेतान् विवर्जयेत्। (Ca.Ci.28/52-53)

Muhurvega (frequent paroxysms) is the commonest feature in case of Ardita, Antarāyāma, Bahirāyāma, Hanugraha, Ākṣepaka and Daṇḍaka. If the frequent paroxysm disappears, the person becomes Svastha (healthy). If these paroxysms do not disappear, then the person gets afflicted with Pīḍana (pain) of the respective diseases. Such person should not be treated by the physician as it is Asādhya (incurable).

Ācārya Carakokta and Suśrutokta Ardita

Ācārya Suśruta described the disease Ardita as one which occurs at only half side of the face. Whereas Ācārya Caraka described that Ardita may occur at half side of the body or half side of the face in paroxysms. And also Ācārya Cakrapāṇi while commenting on this stated the difference between the Ardita and Ardhāṅga (Pakṣāghāta) that Ardita occurs in paroxysm and is not present at all times, whereas Ardhāṅga (Pakṣāghāta) presents at all times.

Ardita Bheda Lakṣaṇās

◉ वातात्पित्तात्कफाच्चस्यात्त्रिविधं तत्समासतः । लालास्रावो व्यथा कम्पः स्फुरणं हनुवाग्ग्रहः ॥
ओष्ठयोः श्वयथुः शूलं चार्दिते वातजे भवेत् ॥ पीतमास्यं ज्वरस्तृष्णापित्तजे मोहधूपते ।
गण्डे शिरसि मन्यायां शोथ स्तम्भः कफात्मके ॥ (B.P.M.24/64-66)

Ardita is classified in to three types namely Vātaja, Pittaja and Kaphaja. The characteristic features are

◈ **Vātaja Ardita:** Lālāsrāva (salivation), Vyathā (throbbing pain), Kampa (tremors), Sphuraṇa (twitching), Hanugraha (stiffness of jaw), Vākgraha (obstructed speech), Oṣṭha svayathu (swelling of lips) and Śūla (sharp pain)

- ◈ **Pittaja Ardita:** Pītamāsya (yellowish discoloration of oral cavity), Jvara (fever), Tṛṣṇā (thirst), Moha (delusion) and Dhūpate (feeling as if smoke is emitting)
- ◈ **Kaphaja Ardita:** Gaṇḍe śirasi manyāyāṃ śotha (swelling over the cheek, head and nape of the neck) associated with Stambha (stiffness).

Sādhyāsādhyata of Ardita

- ◉ क्षीणस्यानिमिषाक्षस्य प्रसक्तं सक्तभाषिणः । न सिध्यत्यर्दितं बाढं त्रिवर्षं वेपनस्य च ॥ (Su.Ni.1/73)

If the patient of Ardita, becomes Kṣīṇa (emaciated) and suffers from Animiṣākṣasya (absence of blinking of an eye), Prasaktaṃ saktabhāṣiṇa (constant unclear speech), Vepana (associated with tremors) and Bādhaṃ trivarṣa (suffering more than three years) should be considered as Asādhya (incurable).

Ardita can be predominantly characterised by conditions like facial palsy, Transient ischemic attack (TIA).

PAKṢĀGHĀTA OR PAKṢĀVADHA, EKĀṄGA ROGA AND SARVĀṄGA ROGA

Nirūkti and Paribhāṣa

The term Pakṣāghāta is a Sanskrit compound consisting of the terms Pakṣa and Āghāta. Pakṣa means flank or side or half of anything and Āghāta means blow or strike or injury. Thus, Pakṣāghāta literally translates to a blow to one side or "injury to one side.

- ◉ पक्षस्य देहाद्धस्य घातं विनाशनं यस्मात् यत्र वा । (SKD)

That which causes Pakṣasya dehāddasya ghāta (devastation of one side of the body) or Vināśana (destruction or annihilation of one side of the body) is called Pakṣāghāta.

- ◉ पक्षाघात एकाङ्गविकारः । एकपक्षविघातमित्युक्तत्वात् ॥ (Āḍ.Śā.P.7/105-114)

Pakṣāghāta is Ekāṅgavikāra (a disorder affecting one part of the body), as it is said to be the Ekapakṣavighāta (destruction or impairment of one side).

Saṃprāpti of Pakṣāghāta

- ◉ अधोगमाः सतिर्यग्गा धमनीरूर्ध्वदेहगाः । यदा प्रकुपितोऽत्यर्थं मातरिश्वा प्रपद्यते ॥
  तदाऽन्यतरपक्षस्य सन्धिबन्धान् विमोक्षयन् । हन्ति पक्षं तमाहुर्हि पक्षाघातं भिषग्वराः ॥
  यस्य कृत्स्नं शरीरार्धमकर्मण्यमचेतनम् । ततः पतत्यसून् वाऽपि त्यजत्यनिलपीडितः । (Su.Ni.1/60-62)

The excessively aggravated Vāta enters the Dhamanis (arteries) and it spreads upwards, side-wards or downwards in the body and Vimokṣayan (liberates) the Sandhi bandhā and leads to the disease Pakṣāghata on either side of the body. In which the affected side becomes Akarmaṇya (inaction) with Acetana (loss of sensation) and Patatyasūn (falls down suddenly) or Tyajatya (even expires).

- ◉ गृहीत्वाऽर्धं तनोर्वायुः सिराः स्नायूर्विशोष्य च॥ पक्षमन्यतरं हन्ति सन्धिबन्धान् विमोक्षयन्।
  कृत्स्नोऽर्धकायस्तस्य स्यादकर्मण्यो विचेतनः॥ एकाङ्गरोगं तं केचिदन्ये पक्षवधं विदुः। (A.H.Ni.15/38-40)

The aggravated Vāta affects half of the body, causing Sirāḥ snāyūviśoṣaṇa (dryness in the veins and ligaments) and leading to the Pakṣamanyataraṃ hanti (destruction of half of the body). This is accomplished by Sandhibandhān vimokṣayan (loosening the joints), resulting in Akarmaṇyo Vicetanaḥ Ardhakāya (inactive and senseless half of the body). This condition is also known as Pakṣāvadha or Ekāṅgaroga.

- ◉ हत्वैकं मारुतः पक्षं दक्षिणं वाममेव वा॥ कुर्याच्चेष्टानिवृत्तिं हि रुजं वाक्स्तम्भमेव च।
  गृहीत्वाऽर्धं शरीरस्य सिराः स्नायूर्विशोष्य च॥ पादं सङ्कोचयत्येकं हस्तं वा तोदशूलकृत्।
  एकाङ्गरोगं तं विद्यात् सर्वाङ्गं सर्वदेहजम्॥ (Ca.Ci.28/53-55)

The aggravated Vāta afflicts either Vāma (left) or Dakṣiṇa pakṣa (right half) of the body and does Śoṣana of Sirā snāyu (dries up the blood vessels and ligaments) of the affected side of the body and produces Ceṣṭānivṛtti (loss of movements), Ruja (pain) and Vākstambha (obstruction of speech). This is known as Pakṣāvadha or Ekāpakṣāvadha.

When aggravated Vāta dries up the Sirā (blood vessels) and Snāyu (ligaments) of one leg or one hand producing Saṅkoca (contraction) of Pāda (leg) or Hasta (hand) associated with Toda (pricking pain) and Śūla (piercing type of pain), it is known as Ekāṅgaroga. And if the aggravated Vāta pervades entire body then it is known as Sarvāṅga roga.

Bheda of Pakṣāghāta

- ◉ दाहसन्तापमूर्छाः स्युर्वायौ पित्तसमन्विते । शैत्यशोथगुरुत्वानि तस्मिन्नेव कफान्विते ॥ (M.N.22/42)

Ācārya Mādhavakara classified Pakṣāghāta into two types based on the associated doṣas, i.e. Pittānvita and Kaphānvita. The characteristic features of **Pittānvita Pakṣāghāta** are Dāha (burning sensation), Santāpa (distress) and Mūrccha (fainting) and the features of **Kaphānvita Pakṣāghāta** are Śaitya (coldness), Śotha (swelling) and Gurutva (heaviness).

Sādhyāsādhyata of Pakṣāghāta

- ◉ शुद्धवातहतं पक्षं कृच्छ्रसाध्यतमं विदुः । साध्यमन्येन संसृष्टमसाध्यं क्षयहेतुकम् ॥
  (गर्भिणीसूतिकाबालवृद्धक्षीणेष्वसृक्स्रुते । पक्षाघातं परिहरेत् वेदनारहितो यदि) ॥ (M.N.22/43)

If the disease Pakṣāghāta occurs with the aggravation of Vāta alone, then it is Kṛcchra sādhya (difficult to cure) and if it occurs in association with Pitta and Kapha, then it is Sādhya (curable) and if it occurs due to Dhātukṣaya (diminution of the dhātus), then it is Asādhya (incurable). Pakṣāghāta occurs in Garbhiṇī (pregnant women), Sūtikā (women in puerperium), Bāla (children), Vṛddha (old age), Kṣīṇa (emaciated), Asṛk Kṣaya (who have lost blood), and Vedanārahito (devoid of pain). It should be rejected for treatment as it is incurable.

Pakṣāghāta, Ekāṅga roga and Sarvāṅga roga can be predominantly characterised by conditions like hemiplegia, monoplegia and quadriplegia respectively.

KAMPAVĀTA / VEPATHU

- ◉ सर्वाङ्गकम्पः शिरसो वायुवेपथुसंज्ञकः । (Va.Sa.Ni.28/154)

In Vepathu, the aggravated Vāta produces Sarvāṅga kampaḥ śiraso (tremors all over the body including the head).

- ◉ करपादतले कंपो देहभ्रमणदुःखिते । निद्राभङ्गो मतिः क्षिणा कंपवातस्य लक्षणम् ॥ (Ba.Rā.6/128)

Karapādatala kaṃpa (tremors in the palm and sole), Dehabhramaṇat (unsteadiness of the body), Nidrābaṅga (loss of sleep) and Matiḥkṣīṇā (reduced intelligence) are the features of Kaṃpavāta.

Kampavāta / Vepathu can be predominantly characterised by conditions like Parkinsonism.

GṚDHRASĪ

- ◉ स्फिक्पूर्वा कटिपृष्ठोरुजानुजङ्घापदं क्रमात्। गृध्रसी स्तम्भरुक्तोदैर्गृह्णाति स्पन्दते मुहुः॥ वाताद्वातकफात्तन्द्रागौरवारोचकान्विता। (Ca.Ci.28/56-57)

In Gṛdhrasī, the aggravated Vāta produces Stambha (stiffness), Ruja (pain), Toda (pricking pain) and Spandate Muhuḥ (frequent twitching) in the Sphik (hip) in the beginning and gradually descends downwards to Kati (waist), Pṛṣtha (back), Ūru (thigh), Jānu (knee), Jaṅga (calf region) and Pada (foot). If the disease is due to Vāta and Kapha, then it produces Tandrā (drowsiness), Gaurava (heaviness), and Arocaka (anorexia) along with the above said features.

- ◉ पार्ष्णिप्रत्यङ्गुलीनां तु कण्डरा याऽनिलार्दिता । सक्थ्नः क्षेपं निगृह्णीयाद्गृध्रसीति हि सा स्मृता ॥ (Su.Ni.1/74)
- ◉ पार्ष्णिं प्रत्यङ्गुलीनां या कण्डरा मारुतार्दिता। सक्थ्युत्क्षेपं निगृह्णाति गृध्रसीं तां प्रचक्षते॥ (A.H.Ni.15/54)

In Gṛdhrasī, the aggravated Vāta afflicts the Kaṇḍarā (tendons) of the Pārṣṇi (heel) and Pratyaṅgulīnāṃ (all the toes) and produces Sakthyutkṣepa nigṛhṇāti (restraining of lifting of the thighs). Ḍalhaṇa while commenting on this, he stated that Gṛdhrasī is also known as Randhiṇī in common language. Āḍhamalla stated that, Gṛdhrasī is popularly known as Rādhinā in common language.

Gṛdhrasī can be predominantly characterised by conditions like sciatica, lumbar spondylolysis, spondylolisthesis with radiculopathy.

VIŚVĀCĪ

- ◉ गृध्रसीसदृशीमेकबाहुगतां विश्वाचीमाह । (Ḍa.Su.Ni.1/75)

The condition that resembles Gṛdhrasī but affects Ekabāhu (one arm) is known as Viśvācī.

- ◉ तलप्रत्यङ्गुलीनां तु कण्डरा बाहुपृष्ठतः । बाह्वोः कर्मक्षयकरी विश्वाचीति हि सा स्मृता ॥ (Su.Ni.1/75)
- ◉ तलं प्रत्यङ्गुलीनां या कण्डरा बाहुपृष्ठतः। बाहुचेष्टापहरणी विश्वाची नाम सा स्मृता॥ (A.H.Ni.15/44)

In Viśvācī, the aggravated Vāta afflicts the Kaṇḍarā (tendons) of the Tala (palm), Pratyaṅgulīnāṃ (all the fingers) and Bāhupṛṣṭhataḥ (back of the arm) and produces Bāhuceṣṭāpaharaṇī (loss of movement of the arm) or Bāhvoḥ karmakṣaya (loss of function of the arm)

Viśvācī can be predominantly characterised by conditions like cervical spondylolysis, spondylolisthesis with radiculopathy, brachial neuralgia, thoracic outlet syndrome, erb's paralysis

Differentiate Gṛdhrasī and Viśvācī

Lakṣaṇās	Gṛdhrasī	Viśvācī
Samprāpti and Lakṣaṇa	The aggravated Vāta afflicts the Kaṇḍarā of the Pārṣṇi and Pratyaṅgulīnāṃ and produces Sakthyutkṣepa nigṛhṇāti.	The aggravated Vāta afflicts the Kaṇḍarā of the Tala, Pratyaṅgulīnāṃ and Bāhupṛṣṭhataḥ and produces Bāhuceṣṭāpaharaṇī or Bāhvoḥ karmakṣaya
Sthāna	Affects the lower extremities	Affects the upper extremities
Bheda	Vātaja and Vātakaphaja	Vātaja and Vātakaphaja (as it resembles Gṛdhrasī – according to Ḍalhaṇa)
Modern parlance	Sciatica, Lumbar spondylolysis, Spondylolisthesis with radiculopathy.	Cervical spondylolysis, Spondylolisthesis with radiculopathy, Brachial neuralgia, Thoracic outlet syndrome, Erb's paralysis

KHALLĪ

- ◉ खल्ली तु पादजङ्घोरुकरमूलावमोटनी॥ (Ca.Ci.28/57)
- ◉ विश्वाची गृध्रसी चोक्ता खल्ली तीव्ररुजान्विते। (A.H.Ni.15/55)

In Khallī, the aggravated Vāta produces twisting pain of the Pāda (feet), Jaṅga (calf region), Ūru (thigh) and Karamūlā (shoulders). Vāgbhaṭa described that the Viśvācī and Gṛdhrasī accompanied with Tīvraruja (severe pain) is called Khallī.

Khallī can be predominantly characterised by conditions like muscle cramps (charley horse), carpopedal spasm, spondylolysis with radiculopathy (both cervical and lumbar together)

KHAÑJA AND PAṄGU

- ◉ वायुः कट्यां स्थितः सक्थ्नः कण्डरामाक्षिपेद्यदा । खञ्जस्तदा भवेज्जन्तुः, पङ्गुः सक्थ्नोर्द्वयोर्वधात् ॥ (Su.Ni.1/77)
- ◉ वायुः कट्यां स्थितः सक्थ्नः कण्डरामाक्षिपेद्यदा। तदा खञ्जो भवेज्जन्तुः पङ्गुः सक्थ्नोर्द्वयोरपि॥ (A.H.Ni.15/45)

The aggravated Vāta gets localized in the Kaṭi (waist) and afflicts the Kaṇḍara (tendon) of the Sakthi (thigh) and produces the disease Khañja. When it afflicts the Kaṇḍara (tendon) of the Sakthyanodvaya (both the thighs), it is known as Paṅgu.

Khañja can be predominantly characterised by conditions like crural monoplegia; whereas, Paṅgu can be predominantly characterised by conditions like crural diplegia, paraplegia

Differentiate Gṛdhrasī and Khañja

Lakṣaṇās	Gṛdhrasī	Khañja
Samprāpti and Lakṣaṇa	The aggravated Vāta afflicts the Kaṇḍarā of the Pārṣṇi and Pratyaṅgulīnāṃ and produces Sakthyutkṣepa nigrhṇāti	The aggravated Vāta gets localized in the Kaṭi and afflicts the Kaṇḍara of the Sakthi and produces Khañja
Sthāna	Either affects the one or both lower extremities	Affects one lower extremities
Bheda	Vātaja and Vātakaphaja	No types
Modern parlance	Sciatica, Lumbar spondylolysis, Spondylolisthesis with radiculopathy	Crural monoplegia

Short Answer questions

1. Describe Apatānaka
2. Explain Gṛdhrasī and Viśvācī
3. Explain Antarāyāma and Bahirāyāma
4. Differntiate Gṛdhrasī and Khañja

Long Answer questions

1. Explain Nirūkti, Samprāpti, Pūrvarūpa, Bheda, Lakṣanās and Sādhyāsādhyata of Ākṣepaka
2. Explain the Nidāna, Samprāpti, Pūrvarūpa, Bheda, Lakṣanās and Sādhyāsādhyata of Ardita.
3. Explain the Nirūkti, Samprāpti, Pūrvarūpa, Bheda, Lakṣanās and Sādhyāsādhyata of Pakṣāghāta

◆

TOPIC 42

Common Neurologic and Spine Disorders

Introduction

Neurological disorders (ND) encompass conditions affecting the central or peripheral nervous system, including the brain, spinal cord, cranial nerves, peripheral nerves, nerve roots, vegetative nervous system, neuro-muscular junction, and muscles. Common neurological disorders comprise Parkinson's disease, stroke, transverse myelitis, epilepsy, multiple sclerosis, Alzheimer's disease, and other dementias. Spinal disorders comprise an array of conditions impacting the structure or function of the vertebral column, spinal cord, or surrounding tissues. Common spinal disorders include lumbago, sciatica syndrome, brachial neuralgia, cervical and lumbar spondylosis, among others.

PARKINSON'S DISEASE (PD)

Introduction

Parkinson's disease was first described by James Parkinson in 1817. The disease mainly affects substantia nigra, which is located in the mid brain that helps in controlling balance and movement. The incidence of parkinson's disease is seen higher among men than women and based on the age of onset it is classified in to three types namely juvenile onset parkinson's disease (less than 20 years of age), late onset parkinson's disease (above 60 years of age) and young onset parkinson's disease (less than 40 – 50 years of age). Sporadic parkinson's disease (with no family history) cause is unknown and about 10 to 15% of patients have genetic predisposition.

Pathophysiology:

Parkinson's disease is characterized by the degeneration of neurons in the substantia nigra, a midbrain region responsible for the production of dopamine, a critical neurotransmitter for motor regulation. The diminishing dopamine levels due to neuronal death lead to the distinctive motor symptoms associated with Parkinson's disease. Additionally, individuals with Parkinson's disease exhibit Lewy bodies, abnormal protein aggregates containing alpha-synuclein, a misfolding and aggregating protein contributing to neuronal degeneration. The loss of dopamine significantly impacts the basal ganglia, a cluster of brain nuclei crucial for motor control, disrupting the equilibrium between excitatory and inhibitory pathways and giving rise to the motor symptoms of Parkinson's disease.

Clinical features:

Bradykinesia (slowness of movement), increased tone (rigidity), tremor at rest (described as pill rolling tremor) and postural instability are the early features of parkinson's disease. Other features include masked facies (hypomimia or loss of facial expressions), decreased arm swing while walking, micro graphia, slurred speech, hyposmia, anxiety, depression, apathy, constipation, sleep disturbance, salivation, weight loss and sexual problems. Vague feelings of weakness and fatigue, in-coordination, aching, and discomfort may also be the indication of onset of the disease.

Diagnosis:

Diagnosis is based on the clinical features, neurological examination and imaging studies. Clinically, presence of bradykinesia along with any of the three other cardinal features of parkinson's disease i.e. resting tremor, rigidity and postural instability. Physical examination reveals presence of dysdiadochokinesia (impaired ability to perform rapid, alternating movements), rigidity (cogwheel or lead pipe) and festinating gait, positive glabellar tap sign and positive extensor plantar responses (Babinski sign). Usually imaging studies such as CT and MRI scan are not required but it may show the degree of brain atrophy in elder patients and PET (positron emitted tomography) scan helps to diagnose the atypical form of parkinsonism.

STROKE

Introduction

Stroke is also known as Cerebro Vascular Accident (CVA) or brain attack, is a heterogeneous group of disorders involving sudden, focal interruption of cerebral blood flow that causes neurologic deficit. Stroke is categorized into mainly 2 types, namely, ischemic and hemorrhagic. Ischemic strokes occur when there is a blockage in a blood vessel, typically resulting from thrombosis or embolism. In contrast, hemorrhagic strokes occur when a blood vessel ruptures, resulting from vascular rupture such as subarachnoid hemorrhage and intracerebral hemorrhage

Stroke ranks as the second leading cause of death worldwide and is a major contributor to disability. Ischemic strokes account for approximately 62% of all strokes, followed by intracerebral hemorrhage at 28% and subarachnoid hemorrhage at 10%. Although ischemic strokes are more prevalent, haemorrhagic strokes result in more fatalities and lost disability-adjusted life-years. Both men and women worldwide face an approximate lifetime risk of stroke of 25% starting from age 25.

Ischemic stroke

Ischemic stroke is primarily caused by large vessel disease, small vessel disease (lacunar), and cardioembolism. Large vessel disease encompasses conditions such as atherosclerosis, arterial dissection, and artery-to-artery embolism. Lacunar strokes, predominantly caused by small vessel diseases, are commonly associated with lipohyalinosis and atherosclerosis. Cardioembolism can stem from various sources, including arrhythmia, valvular heart disease, bioprosthetic and mechanical heart valves, and cardiomyopathy. Ischemic stroke is primarily associated with risk factors such as advanced age, hypertension, diabetes, hyperlipidemia, cigarette smoking, arrhythmia, and cardiac disease.

Ischemic stroke is characterized by inadequate blood supply to a specific area of brain tissue, resulting in irreversible damage to the core area and potential recovery in the surrounding penumbra if early reperfusion is achieved. Diminished blood flow leads to disruptions in adenosine triphosphate (ATP) production, culminating in cellular death. Microglia plays a crucial role in the inflammatory response by releasing both proinflammatory cytokines such as reactive oxygen species (ROS), nitric oxide (NO), interleukin-1β, and tumor necrosis factor-α, as well as anti-inflammatory cytokines and neurotrophic factors, including brain-derived neurotrophic factor, glial cell-line-derived neurotrophic factor, and essential fibroblast growth factor. This sequence ultimately culminates in the loss of neurons and supporting structures.

Hemorrhagic Stroke

Hemorrhagic strokes are divided into intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH). Intracerebral hemorrhage (ICH) is typically caused by the rupture of small arteries due to conditions such as hypertensive vasculopathy and cerebral amyloid angiopathy (CAA), while subarachnoid hemorrhage (SAH) can be caused by a ruptured aneurysm or other factors. Risk factors for both include advancing age, hypertension, smoking, and excessive alcohol consumption.

When small arteries rupture due to various factors, the primary injury mechanisms in intracerebral hemorrhage (ICH) involve the expanding hematoma's mass effect and perihematomal edema, leading to elevated intracranial pressure (ICP) and potential ischemic injury. Patients are also at risk of experiencing intraventricular hemorrhage (IVH) and herniation.

A ruptured cerebral aneurysm is the primary cause of subarachnoid hemorrhage (SAH). However, brain injury from a cerebral aneurysm can occur without a rupture, as compressive forces can damage local brain tissue and compromise blood supply. When an aneurysm ruptures, arterial blood is released into the subarachnoid space, increasing intracranial pressure (ICP) and potentially causing secondary brain injury due to various factors such as intracerebral hemorrhage (ICH), intraventricular hemorrhage (IVH), intracranial pressure (ICP), hydrocephalus, subdural hematoma, or delayed cerebral ischemia (DCI).

Clinical features:

Stroke symptoms typically occur suddenly and may include numbness, weakness in the limbs or face, difficulty speaking, confusion, visual disturbances, dizziness, loss of balance, and headache. An anterior circulation stroke typically causes symptoms on one side of the body. A posterior circulation stroke can cause symptoms on one side or both sides of the body and is more likely to affect consciousness, especially when the basilar artery is involved. The specific symptoms experienced depend on the location of the infarction in the brain.

- **Anterior cerebral artery infarction** may cause contralateral hemiparesis (more pronounced in the leg), urinary incontinence, apathy, confusion, poor judgment, mutism, grasp reflex, and gait apraxia.
- **Middle cerebral artery infarction** may lead to contralateral hemiparesis (more severe in the arm and face than in the leg), difficulty speaking, loss of sensation on one side of the body, contralateral visual field loss, aphasia (if the dominant hemisphere is affected), or apraxia and sensory neglect (if the non-dominant hemisphere is affected).
- **Posterior cerebral artery infarction** may result in contralateral visual field loss, unilateral cortical blindness, memory loss, unilateral 3rd cranial nerve palsy, or hemiballismus.
- **Ophthalmic artery infarction** (a branch of the internal carotid artery) may cause monocular vision loss (amaurosis).
- **Infarctions in the vertebrobasilar system** can lead to unilateral or bilateral cranial nerve deficits, vertigo, difficulty swallowing, difficulty speaking, double vision, blindness, ataxia, spastic weakness, crossed sensory and motor deficits, impaired consciousness, coma, and in severe cases, death (if the basilar artery is completely blocked), as well as rapid heart rate and fluctuating blood pressure.
- **Lacunar infarcts** typically do not cause cortical deficits but may result in pure motor hemiparesis, pure sensory loss on one side of the body, ataxic hemiparesis, or dysarthria-clumsy hand syndrome.

The symptoms of intracerebral hemorrhage (ICH) encompass sudden onset headaches, loss of consciousness, nausea, vomiting, delirium, and seizures. Extensive hemorrhages have the potential to induce hemiparesis (weakness on one side of the body) or brain stem deficits such as conjugate eye deviation or ophthalmoplegia, stertorous breathing, pinpoint pupils, and coma. Approximately half of patients with extensive hemorrhages do not survive beyond a few days. Conversely, small hemorrhages may result in focal deficits without impacting consciousness and can bear resemblance to an ischemic stroke.

The symptoms of subarachnoid hemorrhage (SAH) includes severe headache often described as the worst ever experienced. Loss of consciousness may occur immediately or after several hours. Rapid, irreversible neurological deficits may develop within minutes to hours. Patients may experience impaired mental state, restlessness, and seizures. Initial absence of neck stiffness is common, but chemical meningitis may cause moderate to marked meningismus, vomiting, and irregular heart or respiratory rates within 24 hours. Fever, persistent headaches, and confusion are common in the first 5 to 10 days. Secondary hydrocephalus may cause enduring symptoms, and re-bleeding may lead to recurrent or new symptoms.

Complications

The potential complications following a stroke encompass sleep disturbances, confusion, depression, urinary incontinence, atelectasis (collapse of part of the lung), pneumonia, and dysphagia (difficulty swallowing), which can precipitate aspiration, dehydration, or malnutrition. Prolonged immobility can induce thromboembolic events like deep vein thrombosis, physical deconditioning, sarcopenia (loss of muscle mass), urinary tract infections, pressure ulcers, and joint contractures. Furthermore, diminished daily functionality, encompassing ambulation, vision, sensory perception, cognition, and verbal communication, may ensue.

Diagnosis

Diagnosis is based on clinical evaluation, imaging studies. Imaging studies such as Computed Tomography (CT) helps to quickly identify whether the stroke is ischemic or hemorrhagic by providing detailed images of the brain. Magnetic Resonance Imaging (MRI) provides more detailed images of brain tissue and can detect smaller or deeper brain injuries.

BELL'S PALSY

Introduction

Bell's palsy, also referred to as Facial nerve palsy, denotes an acute onset of unilateral peripheral paralysis of the facial nerve (seventh cranial nerve) of unknown etiology. It is characterized by facial paresis affecting the upper and lower face on one side. Globally, Bell's

palsy affects approximately 25-35 individuals per 100,000 annually and represents 60-70% of all cases of unilateral facial paralysis. In India, the estimated annual incidence of Bell's palsy ranges from 11.5 to 53.3 cases per 100,000 individuals. This condition affects both genders equally and is most prevalent in the age group of 21-40 years. The recurrence rate ranges from 8% to 12%. Notably, even without treatment, 70% of patients experience complete resolution. Risk factors include diabetes, pregnancy, preeclampsia, obesity, and hypertension.

Pathophysiology

Bell's palsy, a condition involving the seventh cranial nerve, results in facial muscle dysfunction. This nerve, responsible for controlling facial expression, traverses a narrow, bony canal in the skull. Viral infections, particularly by Herpes simplex virus (HSV), Varicella-zoster virus (VZV), Epstein-Barr virus (EBV), cytomegalovirus (CMV), and adenovirus, are frequently associated with Bell's palsy. The precise pathophysiological mechanism remains incompletely elucidated; however, it is postulated that viral infections induce an inflammatory response, leading to oedema and ischemia of the facial nerve. Consequently, inflammation may initiate demyelination of the facial nerve, disrupting nerve signal transmission.

Clinical features:

Pain behind the ear is often an early sign of idiopathic facial nerve palsy, which is followed by facial weakness. The weakness usually develops rapidly, reaching its maximum within 48 to 72 hours. Patients may feel numbness or heaviness in the affected side of the face. The face becomes flat and loses its ability to show expression, such as wrinkling the forehead, blinking, and grimacing. In severe cases, the eye on the affected side may not close (ptosis), leading to irritation of the conjunctiva and desiccation of the cornea. While sensory evaluation yields normal results, tenderness may be elicited upon palpation of the external acoustic meatus and a localized region posterior to the ear (over the mastoid). Proximity of the nerve lesion to the geniculate ganglion may further impact salivation and gustatory perception. Lacrimation may be compromised, often accompanied by the co-occurrence of hyperacusis (increased sensitivity to loud sounds).

Diagnosis

The diagnosis relies on clinical evaluation. During examination, the absence of specific facial movements, such as wrinkling of the forehead, the ability to tightly close the eyes, puffing of the face, symmetry of smile, screwing up of eyes, and wrinkling of the nose on the affected side, is carefully assessed. The House-Brackmann Facial Nerve Grading System provides a standardized method for delineating the degree of facial nerve weakness.

Grade	Description	Characteristics
1	Normal	Normal facial function in all areas
2	Mild dysfunction	Slight weakness noticeable on close inspection; may have very slight synkinesis
3	Moderate dysfunction	Obvious, but not disfiguring, difference between two sides; noticeable, but not severe, synkinesis, contracture, or hemifacial spasm; complete eye closure with effort
4	Moderately severe dysfunction	Obvious weakness or disfiguring asymmetry; normal symmetry and tone at rest; incomplete eye closure
5	Severe dysfunction	Only barely perceptible motion; asymmetry at rest
6	Total paralysis	No movement

MOTOR NEURON DISEASE (MND)

Introduction

Motor neuron disease (MND) is a progressive neurodegenerative condition caused by loss of upper and lower motor neurons in the spinal cord, medulla and motor cortex. The incidence is higher in male than female, in the age group between 40 – 60 years. The exact cause is undetermined, but research studies suggested that there is involvement of multiple genes and environmental factors such as exposure to virus and chemicals, extreme physical activity, physical trauma including electric shock, cigarette smoking and high dietary fat intake.

Motor neuron disease is classified in to

- Progressive muscular atrophy (involvement of lower motor neurons (LMN))
- Primary lateral sclerosis (involvement of upper motor neurons (UMN))
- Progressive bulbar palsy (involvement of cranial nerves predominantly)
- Amyotrophic lateral sclerosis (combined involvement of upper and lower motor neurons)

Progressive muscular atrophy

Progressive muscular atrophy is described by Aran in 1850 and it is a progressive degeneration of lower motor neurons. This condition is characterised by weakness and wasting of muscles of the hand and progress to second hand and becomes generalised (including lower limb, trunk and bulbar muscles) over a period of 3-5 years. Other features include fasciculations, clumsiness of the hand and tendon reflexes are preserved or exaggerated. Bulbar and respiratory involvement eventually develops in later stages. Subsequently, some patients may also develop signs of upper motor neurons involvement such as hyper-reflexia and spasticity. In those cases, the diagnosis is changed to Amyotrophic lateral sclerosis (ALS).

Primary lateral sclerosis

Primary lateral sclerosis is uncommon form of motor neuron disease and it is progressive dysfunction of upper motor neurons. This condition is characterised by weakness and spasticity of leg and progress to second leg and becomes generalised (including upper limb, trunk and bulbar muscles) over a period of 4-6 years. Other features include stiffness, clumsiness, poor coordination, balance difficulties, dysarthria, dysphagia, emotional lability (unprovoked laughing or crying), bladder dysfunction, abnormal eye movements and hyper-reflexia. Subsequently, some patients may also develop signs of lower motor neurons involvement such as fasciculations and muscle wasting. Diagnosis needs to be revised to ALS in those cases.

Progressive bulbar palsy (Progressive bulbar atrophy)

Progressive bulbar palsy is the most serious form of motor neuron disease, because it ends the life within a period of 1 year. Progressive bulbar palsy is caused by bilateral lower motor lesion affecting the cranial nerves (especially IX to XII of their nuclei in the oblongata), leading to weakness and wasting of the muscles of the tongue, jaw and face. Fasciculation of tongue is most marked. Other features include weak palatal movement, flaccid dysarthria (nasal speech) and difficulty in swallowing due to progressive involvement of the palatal, pharyngeal and laryngeal muscles. Progressive dysphagia leads to emaciation. Gag reflex and jaw jerk are absent.

Pseudo bulbar palsy (Spastic or Supranuclear bulbar palsy)

Pseudo bulbar palsy is due to an upper motor neuron lesion caused by bilateral disturbance of the corticobulbar tracts and it is characterised by muscle spasticity of tongue, jaw, pharynx and face resulting in difficulty in chewing and swallowing, slurred speech, drooling and hoarseness of voice. Emotional lability (unprovoked laughing or crying) is common. Immobile and inability to protrude the tongue from the mouth and expressionless face occurs due to progressive weakness in tongue and facial muscles respectively. Gag reflex is increased and jaw jerk is exaggerated.

Amyotrophic lateral sclerosis (ALS)

Amyotrophic lateral sclerosis (ALS) is the most common form of progressive motor neuron disease and it is also known as Lou Gehrig Disease and Charcot Syndrome. This condition is characterised by weakness and wasting of the muscles in the upper limbs and then spreads to other parts of the body associated with fasciculation. Other symptoms include spasticity, cramps, sleep disturbance, subtle cognitive and behavioural dysfunction. As the disease progress, bulbar symptoms like dysarthria, dysphonia and dysphagia may be present in most of the cases. Advanced cases may develop emotional impairment and urinary retention or incontinence. Death occurs due to respiratory failure and patient survival is not more than 4-5 years after onset of disease.

Physical examination reveals combination of lower and upper motor neuron signs i.e. some muscles show evidence of UMN lesion in the form of spasticity and exaggeration of deep tendon reflexes; and also evidence of LMN lesion in the form of fasciculation and atrophy. Affliction of the bulbar muscles gives rise to LMN lesion of the tongue, leading to its atrophy, and UMN lesion of the soft palate and pharynx giving rise to exaggeration of gag reflex and dysphagia. The plantar responses are bilaterally extensor indicating UMN lesion.

Diagnosis:

Diagnosis is based on the Serum creatine kinase, Electromyography, Nerve conduction studies, MRI scan and Transcranial Magnetic Stimulation. Usually Serum creatine kinase levels are normal or mildly elevated. Needle Electromyography (EMG) reveals fibrillation potentials, positive sharp waves and fasciculation potentials usually confirm the typical features of widespread denervation and re-innervation. Nerve conduction studies (NCS) are normal but there may be reduction in amplitude of motor action potentials due to axonal loss. MRI scan of brain and cervical spine helps to rule out absence of structural lesions or injuries which mimics the MND. Transcranial Magnetic Stimulation (TMS) should be carried out at the same time of nerve conduction studies (NCS) to measure the activity of upper motor neurons.

TRANSVERSE MYELITIS

Introduction

Transverse myelitis (TM) is a rare, acquired focal inflammatory disorder that impacts the spinal cord at any level, with the thoracic region being the most commonly affected. It manifests through rapid onset weakness, sensory deficits, and bowel/bladder dysfunction. TM can arise independently or as a consequence of infection and is often linked to other neuro-inflammatory disorders, including acute disseminated encephalomyelitis, multiple sclerosis, neuromyelitis optica spectrum disorder, and acute flaccid myelitis. The primary cause of TM is idiopathic, with infections (such as enteroviruses, West Nile virus, herpes viruses, HIV, human T-cell leukemia virus type 1 (HTLV-1), Zika virus, neuroborreliosis (Lyme), Mycoplasma, and Treponema pallidum) and various autoimmune disorders (comprising multiple sclerosis, neuromyelitis optica spectrum disorder, acute disseminated encephalomyelitis, ankylosing spondylitis, antiphospholipid syndrome, Behçet disease, mixed connective tissue disease, rheumatoid arthritis, sarcoidosis, scleroderma, Sjögren syndrome, and systemic lupus erythematosus) being potential contributing factors.

Transverse myelitis can affect both genders equally, but women are more commonly affected in cases linked with multiple sclerosis. While it has the potential to manifest in individuals across all age groups, its incidence is most prevalent among those aged 10 to 19 and 30 to 39.

Pathophysiology

The primary characteristic of Transverse Myelitis is inflammation, which may manifest in any segment of the spinal cord. This inflammatory process frequently damages the myelin sheath, the protective covering of nerve fibres, thereby disrupting the normal conduction of nerve signals between the spinal cord and the peripheral nervous system. In numerous cases, Transverse Myelitis is considered to be immune-mediated, wherein the immune system erroneously targets the spinal cord, precipitating inflammation and myelin damage. Infections or other autoimmune conditions can incite this immune response. Additionally, certain infections have the potential to directly inflict damage upon the spinal cord tissue, exacerbating the symptoms associated with Transverse Myelitis.

Clinical features:

The interruption of nerve signal transmission can result in a spectrum of symptoms, including motor, sensory, and autonomic dysfunction.

Motor dysfunction:

- Motor deficits encompass rapidly progressing paraparesis, which may initially manifest in the upper extremities with flaccidity followed by spasticity. This is often attributed to damage to white matter structures in the spinal cord.
- Motor symptoms and their severity depend on the level of the spinal cord involved. Upper cervical lesions may affect all four extremities, while lower cervical lesions may cause specific motor signs in different extremities.
- Thoracic lesions are the most common and may lead to motor signs in the lower extremities. Lumbar lesions are less common and affect the lower extremities.

Sensory dysfunction:

- Sensory involvement commonly presents with symptoms such as pain, dysesthesia, and paresthesia at the affected level.
- Sensory symptoms usually correspond to the level of the lesion and may be accompanied by back pain.

Autonomic dysfunction:

- Autonomic manifestations of transverse myelitis include urinary urgency, bladder/bowel incontinence, difficulty or inability to void, bowel constipation, or sexual dysfunction.

- Urinary retention may serve as the initial indicator of myelitis and necessitate further investigation into myelopathy

Diagnosis

Diagnosis of transverse myelitis is typically based on Magnetic resonance imaging (MRI) and analysis of cerebrospinal fluid (CSF). MRI imaging typically indicates cord swelling in the presence of transverse myelitis and can aid in the exclusion of other treatable causes of spinal cord dysfunction, such as spinal cord compression. Furthermore, CSF analysis often reveals an increase in monocytes, slightly elevated protein content, and an elevated IgG index (normal, ≤ 0.85).

EPILEPSY (ORGANIC)

Epilepsy is a group of disorders characterized by recurrent episodes of altered brain function due to abnormal and synchronized electrical activity in the brain. These episodes, known as seizures, can be convulsive or non-convulsive. Organic epilepsy, also referred to as symptomatic epilepsy, denotes seizures arising from identifiable structural or metabolic irregularities within the brain, including brain injuries (trauma, stroke, tumors), infections (meningitis, encephalitis), genetic disorders (Tuberous Sclerosis, Neurofibromatosis), developmental disorders (cortical dysplasia), neurodegenerative diseases (Alzheimer's, Parkinson's), vascular malformations (arteriovenous malformations), brain tumors (benign or malignant), and cerebral vasculitis.

The World Health Organization (WHO) estimated that 65 million people globally and 10-12 million individuals in India live with epilepsy. Symptomatic epileptic seizures are most prevalent among neonates and the elderly. Common triggers for these seizures include fever, hereditary or congenital neurological disorders, birth injuries, inherited or acquired metabolic disorders, cerebral trauma, alcohol withdrawal, tumors, and strokes.

Classification of Seizures

A seizure is classified into generalized onset and focal onset.

- **Generalized onset seizures** arise from a bilateral side of the brain, affecting the whole body and classified into motor and non motor seizure.
  - **Generalized motor seizure** includes tonic-clonic seizures (grand-mal seizure) characterized by abrupt loss of consciousness, stiffens, twitches and jerks of the whole body, bladder and bowel incontinence with biting of the tongue and it usually lasts for two to five minutes.
  - **Generalized non motor seizure** includes absent seizures (petit mal seizure) commonly seen in children and adolescents and characterized by abrupt loss of consciousness for a moment without abnormal movements associated with loss of awareness, staring or inattentive and repeated movements such as lip smacking or blinking of eyes.

- **Focal onset seizures** or partial seizures arise from one part of the brain and it is classified into focal aware seizures (simple) and focal impaired-awareness seizures (complex partial seizures).
  - **Simple focal seizure** is characterized by twitching movements, tingling sensation along with bizarre vision, taste or smell without loss of consciousness.
  - **Complex focal seizures** are characterized by confused, unresponsive, automatic motions such as finger fumbling or lip smacking. Both types of focal onset seizures can spread and affects the entire brain and may cause generalized seizure.

Diagnosis:

Diagnosis is based on medical history, physical examination, electroencephalogram (EEG) and neuro-imaging studies such as CT and MRI. Medical history and complete neurological examination gives the clue for diagnosis. For example, Fever and neck stiffness may be due to meningitis, asymmetry of reflexes or muscle strength may be due to tumor. EEG records the electrical impulses of the brain to detect an epileptiform abnormality. CT and MRI scan is done to identify the structural abnormalities such as brain tumors, abscesses, cortical dysplasia, cerebral venous thrombosis, herpes encephalitis, mesial temporal sclerosis and traumatic gliosis.

LUMBAGO

Introduction

Lumbago is also known as low back pain or low backache, represents the most prevalent musculoskeletal condition, characterized by either dull or sharp pain in the posterior trunk extending from the lower ribcage to the horizontal gluteal crease. Recent studies indicate that approximately 577 million individuals globally experience low back pain, constituting approximately 7.5% of the global population. In India, the estimated prevalence of low back pain ranges from 48% to 66% of the population.

Low back pain can stem from various conditions, including traumatic injuries, degenerative conditions, oncologic issues, infections, inflammatory conditions, metabolic issues, referred pain, postural strain, congenital conditions, and psychiatric factors. Contributory risk factors include age, obesity, smoking, and a sedentary lifestyle.

Pathophysiology:

Low back pain is a complex condition that involves various factors including biomechanics, neurophysiology, and psychosocial aspects. The pathophysiological cascade commences with muscle strain or disc degeneration, leading to inflammation and activation of pain-sensing nerve endings. This can result in increased pain perception. The involvement of pro-inflammatory cytokines, pain mediators, and neurotransmitters like substance P (SP) and serotonin is crucial. As the condition progresses, muscle spasms, ligament strain, and facet joint dysfunction can contribute to chronic pain and disability.

Clinical features:

Low back pain is classified based on the duration

- **Acute low back pain** refers to back pain lasting fewer than 6 weeks.
- **Subacute low back pain** denotes back pain persisting for 6 weeks or more but less than 12 weeks.
- **Chronic low back pain** indicates back pain that hinders the patient from engaging in certain life activities for 12 weeks or more.
- **Recurrent low back pain** describes the occurrence of acute low back pain in a patient who has previously experienced similar episodes, with asymptomatic intervals in between.

In addition, pain may manifest following movements involving lifting, twisting, or **forward-bending.** The onset of symptoms may occur soon after such movements or upon **awakening the following** morning. Symptoms can range from tenderness at specific points to **diffuse pain,** and may or may not exacerbate with particular movements, such as leg raising, **or specific positions,** such as sitting or standing. Red flag signs include sudden severe pain, **trauma or recent** injury, fever, chills, weight loss, numbness, tingling, or weakness in the **lower extremities,** difficulty controlling bowel or bladder function, and abnormal reflexes or **sensory deficits.**

Diagnosis:

Diagnosis is based on clinical evaluation and Imaging studies. During the examination, attention is given to posture, gait, visible deformities, tenderness, spasms, or anomalies in the spine and surrounding musculature. Furthermore, flexibility and mobility of the spine are evaluated, along with the assessment of reflexes, muscle strength, and sensory responses to detect potential nerve involvement.

Imaging studies, such as X-rays, are instrumental in detecting bone abnormalities, fractures, or degenerative changes. Magnetic Resonance Imaging (MRI) yields detailed images of soft tissues, encompassing discs, muscles, and nerves, facilitating the identification of herniated discs, spinal stenosis, or other soft tissue pathologies. Computed Tomography (CT) furnishes detailed cross-sectional images of the spine, aiding in the diagnosis of complicated bone and joint conditions. Electromyography (EMG) quantifies the electrical activity of muscles to detect nerve compression or damage.

SCIATICA SYNDROME

Introduction

Sciatica, or Sciatica Syndrome, is a debilitating condition characterized by pain or paresthesias along the distribution of the sciatic nerve or an associated lumbosacral nerve root. The primary etiologies include a herniated or protruding lumbar intervertebral disc, lumbar spinal stenosis, and spondylolisthesis. Moreover, sciatic symptoms may arise from muscular spasms or inflammation in the lumbar or pelvic region, exerting pressure on a nerve root. In certain instances, the manifestation of sciatica symptoms may be attributed to the presence of a spinal or paraspinal mass, such as malignancy, epidural hematoma, or epidural abscess. The nerve roots most frequently affected are L5-S1, L4-L5, and L3-L4.

The global prevalence of sciatica exhibits significant variability, with estimates ranging from 1.6% to 43% depending on the demographics of the studied population. On average, the prevalence is approximately 18%. The annual incidence of sciatica is estimated to range between 1% and 5%. In India, the prevalence of low back pain, encompassing sciatica cases, ranges from 42% to 83%. While specific data on isolated sciatica cases is less frequently reported, it substantially contributes to the overall burden of low back pain. Moreover, higher prevalence rates were observed among females, rural populations, and individuals engaged in elementary occupations.

Pathophysiology

Sciatica occurs when the sciatic nerve is compressed or irritated, leading to a multifaceted pathophysiological response. This involves mechanical stress on surrounding structures, such as intervertebral discs, facet joints, and the piriformis muscle. Conditions like herniated discs, degenerative disc disease, spinal stenosis, and spondylolisthesis can cause nerve compression, resulting in inflammation and oedema. This amplifies pain perception, while neurotransmitter imbalances contribute to chronic pain. Pro-inflammatory cytokines, pain mediators, and neurotransmitters like substance P and serotonin play significant roles in hormonal changes such as elevated cortisol levels. Risk factors include age, obesity, sedentary lifestyle, genetics, previous injury, and psychosocial factors.

Clinical features:

Patients suffering from sciatica may present with various neurological symptoms, Including radicular pain within the lumbosacral nerve root distribution, sensory impairment or disturbance manifesting as hot and cold sensations, tingling, burning sensations, or numbness in the legs, muscular weakness, reflex impairment, and gait dysfunction.

Sciatica symptoms, such as paresthesias or dysesthesias and edema in the lower extremity, may vary depending on the affected nerve:

- **L4:** Compression or irritation of the L4 nerve may result in pain, tingling, and numbness in the thigh, weakness during leg straightening, and a diminished knee jerk reflex.
- **L5:** Compression or irritation of the L5 nerve may lead to pain, tingling, and numbness extending to the foot and big toes.
- **S1:** Compression or irritation of the S1 nerve may cause pain, tingling, and numbness on the outer part of the foot, weakness when elevating the heel off the ground and standing on tiptoes, and a diminished ankle jerk reflex.

Diagnosis:

Diagnosis is based on clinical examination, imaging studies and electro-diagnostic studies. During the examination, specific tests are conducted to aid in the diagnosis of sciatic nerve pain. The **Straight leg raise (SLR) test** yields a positive result when radiating pain is experienced along the sciatic nerve and below the knee within a range of 30 to 70 degrees of hip flexion. The **Bragard test** is considered positive if symptoms reoccur with ankle dorsiflexion at the conclusion of the SLR. **The Bowstring test** yields a positive if the patient experiences pain when pressure is applied with the thumb on the popliteal fossa over the sciatic nerve (posterior tibial nerve) while the knee is flexed. The **Naffziger test** involves inducing pain through coughing. Additionally, the **Crossed straight leg test**, which mirrors the straight leg raise test but is performed on the asymptomatic leg, is deemed positive if pain is reported in the symptomatic leg when the asymptomatic leg is raised to a 40° angle, indicating a central disc herniation with severe nerve root irritation.

Imaging studies, such as Magnetic Resonance Imaging (MRI) and Computed Tomography (CT), accurately reveal structural abnormalities causing sciatica, including spinal stenosis. Additionally, electrodiagnostic studies serve to substantiate the presence and extent of nerve root compression while excluding conditions that may imitate sciatica, including peroneal nerve palsy, multiple mononeuropathy, or polyneuropathy.

BRACHIAL NEURALGIA

Introduction

Brachial neuralgia, also called brachial neuritis, brachial amyotrophy, Parsonage-Turner syndrome, is characterized by an abrupt onset of excruciating unilateral shoulder pain, followed by flaccid paralysis of shoulder and parascapular muscles several days later. This condition is frequently preceded by a viral illness such as hepatitis E virus, coxsackie viruses, Covid-19 virus, Escherichia coli, Staphylococcus aureus, and Aspergillus), surgery, connective tissue disorders, systemic lupus erythematosus, temporal arteritis, immunizations, pregnancy, and radiation therapy. The affected musculature is predominantly innervated by the C5/6 segmental level, suggesting the involvement of the upper trunk of the brachial plexus or individual nerves derived from the plexus, such as the long thoracic, axillary, suprascapular, and anterior interosseous nerves.

The global incidence of brachial neuritis is estimated to range between 1.6 and 3 cases per 100,000 persons annually. Furthermore, it exhibits a predilection for males over females, with a heightened occurrence among middle-aged individuals, typically around the age of 40.

Pathophysiology

The etiology of brachial neuritis remains incompletely understood, but evidence suggests involvement of an immune-mediated process affecting the brachial plexus directly or through an indirect autoimmune response. Furthermore, in mechanical etiologies, immunological implications arise secondary to compromised blood–nerve barrier integrity, permitting inflammatory mediators to inflict damage upon brachial plexus nerves. Ischemia, stemming from either mechanical or inflammatory causes, is also implicated in the abrupt onset of severe pain in brachial neuritis patients.

Clinical features:

The condition is characterized by acute shoulder pain followed by subsequent muscular weakness. The pain typically presents unilaterally, with a sudden onset and persistent nature, often described as burning, sharp, or throbbing. It may radiate to the neck and forearm due to the involvement of various nerves. Patients may also demonstrate paralysis, sensory deficits, and impaired muscle control in the affected arm or shoulder.

Diagnosis:

Diagnosis is based on clinical examination, imaging studies and electro-diagnostic studies. During a physical examination, it is important to conduct a comprehensive neurological assessment. This should include evaluating the movement of the shoulder blades during abduction and forward movement, as well as testing the strength of the shoulder. One distinctive feature that sets brachial plexus neuritis apart from other conditions is the involvement of muscles innervated by the same peripheral nerve (pathy paresis). Signs of lower motor neuron involvement may become apparent later in the progression of the disease, including symptoms such as reduced muscle tone, absent reflexes, muscle wasting, and muscle twitching, particularly in the upper brachial plexus (at the C5-C7 vertebral levels). Following the acute pain phase, weakness may be accompanied by reduced shoulder abduction and external rotation, indicating the affected deltoid, supraspinatus, and infraspinatus muscles. If the long thoracic nerve is affected, medial scapular winging can occur. Additionally, sensory changes may be present in approximately 78% of patients, manifesting as tingling and reduced sensation in the lateral arm, deltoids, and radial forearm.

Imaging studies such as magnetic resonance imaging (MRI) of the shoulder and spine demonstrates the structural abnormalities and hyperintense signal indicating inflammation or "hourglass" constrictions of the affected nerve. Electro-diagnostic studies such as Nerve conduction indicate diminished amplitudes, while electromyography (EMG) findings suggest acute denervation. Subsequent stages exhibit signs of chronic denervation and decreased movement amplitude.

CERVICAL SPONDYLOSIS

Introduction

Cervical spondylosis, an osteoarthritis condition affecting the cervical spine, is characterized by neck pain that may radiate to the posterior occipital region, shoulders, or arms, and is associated with headaches. The condition predominantly arises from age-related degeneration of intervertebral discs and cervical spinal elements, resulting in the narrowing of the spinal canal and intervertebral foramina. This degeneration can present as axial neck pain, cervical myelopathy, or cervical radiculopathy. The C6-C7 and C5-C6 levels are most commonly affected.

Contributing factors to an accelerated disease process include spinal trauma, congenitally narrow vertebral canal, dystonic cerebral palsy affecting cervical musculature, and participation in specific athletic activities such as rugby, soccer, and horse riding.

In 2017, the global incidence of neck pain, including cervical spondylosis, was estimated at approximately 3551.1 per 100,000 individuals. Notably, cervical spondylosis exhibits a high prevalence in India, impacting over 85% of individuals aged 45 to 60, with an estimated annual impact on 10 million people. This condition affects both genders, with a higher severity observed in females.

Pathophysiology:

The development of cervical spondylosis involves a degenerative process that leads to biomechanical changes in the cervical spine, resulting in compression of neural and vascular structures. This process causes the intervertebral disc to lose hydration, leading to changes in the load distribution along the spine and the formation of bone spurs. These degenerative changes result in the loss of cervical lordosis and movement, as well as a reduction in the diameter of the spinal canal.

Clinical features:

Symptomatic cervical spondylosis typically manifests as one or more of the following three primary clinical syndromes

- **Axial Neck Pain:** Patients commonly report stiffness and pain in the cervical spine, which is most severe in the upright position and alleviated by bed rest. Neck motion, particularly hyperextension and side-bending, typically exacerbates the pain. In cases of upper and lower cervical spine disease, patients may describe experiencing radiating pain into the back of the ear or occiput, or into the superior trapezius or periscapular musculature. Occasionally, patients may present with atypical symptoms of cervical angina, such as jaw pain or chest pain.
- **Cervical Radiculopathy:** Radicular symptoms usually follow a myotomal distribution depending on the involved nerve root(s) and may present as unilateral or bilateral neck pain, arm pain, scapular pain, paresthesia, and arm or hand weakness. Pain is exacerbated by head tilt towards the affected side or by hyperextension and side-bending towards the affected side.
- **Cervical Myelopathy:** This condition typically has an insidious onset with or without neck pain, which is frequently absent. It may initially present with hand weakness and clumsiness, leading to an inability to perform tasks requiring fine motor coordination, such as buttoning a shirt, tying shoelaces, or picking up small objects. Patients often report gait instability and unexplained falls. Urinary symptoms, such as incontinence, are rare and typically manifest late in the disease progression.

Diagnosis:

Diagnosis is based on clinical evaluation and imaging studies. During the examination, a positive **Spurling test** indicates radiating pain down the upper limb with head extension and ipsilateral head rotation to the affected side. A positive **Lhermitte's sign** manifests as an electric shock-like sensation radiating down the spine and into the extremities with cervical flexion. In cases of cervical spondylotic myelopathy, eliciting **Hoffman's sign** involves flicking the patient's distal phalanx of the middle finger and observing reflexive flexion of the thumb and/or index finger.

Imaging studies such as plain x-ray of the cervical spine, reveal osteophyte formation, disc space narrowing, endplate sclerosis, degenerative changes of uncovertebral and facet joints, and calcified/ossified soft tissues. Magnetic Resonance Imaging (MRI) aids in quantifying the extent of nerve and cord compression and identifying pathological changes, such as herniated discs, bony spurs, ligamenta flava hypertrophy, or facet joint arthropathy. Hyperintense spinal cord signal on T2-weighted images may indicate oedema, inflammation, ischemia, myelomalacia, or gliosis. Computed Tomography (CT) unveils intervertebral foraminal stenosis in the setting of uncovertebral or facet hypertrophy, while CT Myelogram demonstrates the location and degree of neural compression.

LUMBAR SPONDYLOSIS

Introduction

Lumbar spondylosis is a chronic and progressive condition characterized by degenerative changes in the lumbar spine. It includes a range of degenerative changes in the intervertebral discs, facet joints, and other spinal structures. These changes include the development of bony spurs (osteophytes), sclerosis of vertebral bodies or endplates, and hypertrophy of ligaments. In severe cases, lumbar spondylosis can lead to the narrowing of the spinal canal or disc space, potentially resulting in disability. However, this constriction primarily affects the nerve roots of the cauda equina rather than the spinal cord because the spinal cord ends at the level of the first or second lumbar vertebra (L1-L2). This condition is commonly associated with ageing and presents symptoms such as lower back pain, stiffness, and reduced mobility. Risk factors, such as ageing, obesity, and a sedentary lifestyle, accelerate this process. Genetic predisposition, trauma, and occupational factors also contribute to the development and progression of lumbar spondylosis.

Globally, an estimated 266 million individuals (3.63% of the population) are affected by degenerative lumbar spine disease (DSD) annually, which includes lumbar spondylosis. The prevalence of low back pain (LBP), often linked to lumbar spondylosis, ranges from 42% to 83%. A systematic review revealed the point prevalence of LBP in India to be 48%, the annual prevalence to be 51%, and the lifetime prevalence to be 66%. These rates are notably higher among females, rural populations, and elementary workers.

Pathophysiology:

Lumbar spondylosis involves degenerative changes in the lumbar spine due to biomechanical and biochemical processes. This includes disc dehydration, proteoglycan loss, facet joint osteoarthritis, and vertebral body sclerosis. As disc degeneration progresses, the annulus fibrosus weakens, leading to herniation and inflammation. This condition also causes muscle spasms, nerve compression, and amplified pain perception. Altered biomechanics and movement patterns perpetuate the degenerative cycle.

Clinical features:

Lumbar spondylosis may manifest as either symptomatic or asymptomatic. Generally, it presents as asymptomatic; however, when symptomatic, it is characterized by sensory or motor dysfunction in the lower extremities and pain radiating towards the hip or legs, particularly sciatica in younger individuals. Advanced degeneration of the lumbar spine can result in spinal cord compression, leading to alterations in sphincter function, such as neurogenic bladder or neurological deficits. Notably, chronic lumbar spondylosis is often associated with neurogenic claudication, a serious condition caused by lumbar spinal stenosis.

Diagnosis:

Diagnosis is based on clinical evaluation and imaging studies. During the examination, it's important to assess the range of motion, identify areas of tenderness, and conduct specific tests. These include the **Straight Leg Raise (SLR) test** to ascertain nerve root irritation, the **Femoral Nerve Tension Test** for assessing femoral nerve irritation and mobility, the **Faber Test (Patrick's Test)** for evaluating hip joint or sacroiliac joint pathology, and **Ely's Test** for examining tightness in the rectus femoris muscle or femoral nerve irritation.

Imaging studies, such as X-rays, reveal structural changes in the vertebrae, including bone spurs (osteophytes) and disc space narrowing. Magnetic Resonance Imaging (MRI) offers detailed images of soft tissues, including discs and nerves, aiding in the detection of herniated discs or spinal stenosis. Computed Tomography provides detailed images of bone structures.

MCQ's

1. Which part of the brain is primarily affected in Parkinson's disease
   a) Hippocampus
   b) Cerebellum
   c) Substantia nigra
   d) Medulla oblongata
2. What is the main neurotransmitter involved in Parkinson's disease
   a) Serotonin
   b) Dopamine
   c) Acetylcholine
   d) GABA
3. Which of the following is NOT a cardinal feature of Parkinson's disease
   a) Bradykinesia
   b) Resting tremor
   c) Rigidity
   d) Hyperreflexia
4. What is another term for stroke
   a) Myocardial Infarction
   b) Cerebrovascular accident
   c) Transient ischemic attack
   d) Epileptic seizure
5. What is the primary cause of subarachnoid haemorrhage (SAH)
   a) Hypertensive vasculopathy
   b) Cerebral amyloid angiopathy
   c) Ruptured cerebral aneurysm
   d) Arterial dissection
6. What is another term for Bell's palsy
   a) Facial nerve palsy
   b) Trigeminal neuralgia
   c) Glossopharyngeal neuralgia
   d) Vestibular neuritis

7. Which cranial nerve is affected in Bell's palsy
a) Fifth cranial nerve
b) Seventh cranial nerve
c) Sixth cranial nerve
d) Eighth cranial nerve

8. Which viral infection is most commonly associated with Bell's palsy
a) Herpes simplex virus
b) Influenza virus
c) Human papillomavirus
d) Hepatitis B virus

9. Which type of motor neuron disease involves only lower motor neurons
a) Primary lateral sclerosis
b) Progressive bulbar palsy
c) Progressive muscular atrophy
d) Amyotrophic lateral sclerosis

10. What is the most common form of motor neuron disease
a) Primary lateral sclerosis
b) Progressive bulbar palsy
c) Progressive muscular atrophy
d) Amyotrophic lateral sclerosis

11. Which motor neuron disease is characterized by emotional lability, such as unprovoked laughing or crying
a) Progressive muscular atrophy
b) Primary lateral sclerosis
c) Progressive bulbar palsy
d) Pseudo bulbar palsy

12. Which cranial nerves are predominantly affected in progressive bulbar palsy
a) Cranial nerves I to IV
b) Cranial nerves V to VIII
c) Cranial nerves IX to XII
d) Cranial nerves XIII to XVI

13. Which region of the spinal cord is most commonly affected by transverse myelitis
a) Cervical
b) Thoracic
c) Lumbar
d) Sacral

14. What is the primary characteristic of transverse myelitis
a) Degeneration of motor neurons
b) Inflammation of the spinal cord
c) Demyelination of peripheral nerves
d) Atrophy of muscle tissue

15. What is the most common type of generalized seizure
a) Tonic seizure
b) Clonic seizure
c) Tonic-clonic seizure
d) Focal seizure

16. What term describes a seizure affecting only one part of the brain
a) Focal seizure
b) Segmental seizure
c) Grand-mal seizure
d) Petit mal seizure

17. What is another term for lumbago
a) Sciatica
b) Low back pain
c) Cervicalgia
d) Thoracic pain

18. Which nerve roots are most frequently affected in sciatica
a) L1-L2
b) L3-L4
c) L5-S1
d) S2-S3

19. Which test is positive when radiating pain is experienced along the sciatic nerve and below the knee within a range of 30 to 70 degrees of hip flexion
a) Bragard test
b) Bowstring test
c) Naffziger test
d) Straight leg raise (SLR) test

20. Which nerve compression may result in pain, tingling, and numbness on the outer part of the foot, and a diminished ankle jerk reflex
a) L4
b) L5
c) S1
d) S2

21. What is another term for brachial neuralgia
a) Trigeminal neuralgia
b) Brachial amyotrophy
c) Carpal tunnel syndrome
d) Thoracic outlet syndrome

22. Which segmental levels are predominantly affected in brachial neuralgia
a) C1-C2
b) C3-C4
c) C5-C6
d) C7-C8

23. Which levels of the cervical spine are most commonly affected by cervical spondylosis
a) C1-C2
b) C3-C4
c) C5-C6
d) C7-T1

24. Which clinical syndrome of cervical spondylosis is characterized by stiffness and pain in the cervical spine, most severe in the upright position
a) Axial Neck Pain
b) Cervical Radiculopathy
c) Cervical Myelopathy
d) Cervical Angina

Answer Keys

1	c	2	b	3	d	4	b	5	c	6	a	7	b	8	a
9	c	10	d	11	d	12	c	13	b	14	b	15	c	16	a
17	b	18	c	19	d	20	c	21	b	22	c	23	c	24	a

Short Answer Questions

1. Explain Parkinson's disease
2. Explain Stroke
3. Explain Bell's palsy
4. Explain Motor neuron disease
5. Explain Transverse myelitis
6. Explain organic epilepsy
7. Describe Lumbago
8. Explain Sciatica syndrome
9. Explain Brachial neuralgia
10. Explain cervical spondylosis
11. Explain lumbar spondylosis

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TOPIC 43

Sandhigatavāta and Asthimajjāgatavāta

SANDHIGATAVĀTA

Sandhigatavāta is one of the Vātavyādhi, where Vāta afflicts the Sandhīs (joints) and produce Śūla (pain) and Śotha (swelling) in the affected joint. Ācārya Caraka, Vāgbhaṭa and Suśruta explained Sandhigatavāta under the Gata vāta. Sandhivāta and Sandhigata Anila are the synonyms of Sandhigatavāta

Sāmānya Nidāna and Saṃprāpti

The Nidāna (causative factors) and Saṃprāpti (pathogenesis) of Sandhigatavāta are not mentioned in the Saṃhitās. Hence the general causative factors and pathogenesis described for Vātavyādhi can be considered for this disease.

◉ रूक्षशीताल्पलघ्वन्नव्यवायातिप्रजागरैः। विषमादुपचाराच्च दोषासृक्स्रवणादति॥ लङ्घनप्लवनात्यध्वव्याया
मातिविचेष्टितैः। धातूनां सङ्क्षयाच्चिन्ताशोकरोगातिकर्षणात्॥ दुःखशय्यासनात् क्रोधाद्दिवास्वप्नाद्भयादपि।
वेगसन्धारणादामादभिघातादभोजनात्॥ मर्माघाताद्गजोष्ट्राश्वशीघ्रयानापतंसनात्। देहे स्रोतांसि रिक्तानि पूरयित्वाऽनिलो
बली॥ करोति विविधान् व्याधीन् सर्वाङ्गैकाङ्गसंश्रितान्। (Ca.Ci.28/15-19)

Due to intake of Rūkṣa (unctuous substances), Śīta (cold substances), Alpa (less in quantity), Laghvanna (light diet); indulging in Ativyavāya (excessive sexual intercourse), Prajāgaraiḥ (remaining awake at night time), Viṣamādupacāra (inappropriate application of therapeutic measures), Doṣāsṛksravaṇāt (excessive elimination of Doṣas including Malas and Rakta), Laṅghana (fasting), Plavana (swimming), Atyadhva (excessive walking), Ativyāyāma (excessive exercise), Aticeṣṭita (excessive physical exertion), Dhātunāṃ saṃkṣayāt (depletion of dhātus), Cintāśokarāgātikarṣaṇāt (excessive emaciation due to worries, grief and diseases), Krodhāt (anger), Duḥkhaśayyāsanāt (sleeping or sitting over uncomfortable beds or seats), Divāsvapnā (sleeping during day time), Bhayāt (fear), Vegasaṃdhāraṇāt (suppression of natural urges), Āmāt (Āma doṣa), Abhighātāt (injury), Abhojanāt (abstinence from food), Marmāghātāt (injuries to vital parts of the body) and Gajoṣṭrāśvaśīghrayānāpatamsanāt (riding over an elephant, camel, horse or fast moving vehicles and falling from the seats on these animals and vehicles) leads to aggravation of Vāta, which fills up the Riktāni deha srotāṃsi (empty channels of the body) and manifests various diseases affecting the Sarvāṅga (entire body) or Ekāṅga (a specific part of the body).

Saṃprāpti Ghaṭaka of Sandhigatavāta

Doṣa	Vāta Kapha
Dūṣya	Asthi, Majjā and Snāyu
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Māṃsa, Medas, Asthi and Majjāvahā
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Sarva Rasāyanīs
Adhiṣṭhāna	Sandhi
Udbhava sthān	Pakvāśaya
Vyakta sthāna	Sandhi
Rogamārga	Madhyama

Sandhigatavāta Lakṣaṇās

- वातपूर्णदृतिस्पर्शः शोथः सन्धिगतेऽनिले। प्रसारणाकुञ्चनयोः प्रवृत्तिश्च सवेदना॥ (Ca.Ci.28/37)
- हन्ति सन्धिगतः सन्धीन् शूलशोफौ करोति च । (Su.Ni.1/28)

When vitiated Vāta is located in the Sandhi (joints), it produces Vātapūrṇādṛtisparśa śotha (swelling appears like an air filled bag) and Prasāraṇākuñjanayo pravṛtti savedanā (appearance of pain during flexion and extension of the joints). Ācārya Suśruta added some other features such as Hanti sandhīn (diminution of the movements at joints), Sandhi śūla (pain in the joints) and Sandhi śopha (swelling of the joints).

- ये वातव्याधयः सन्धीनाश्रयन्ति शनैः शनैः। सद्यो वा शूलशोफाभ्यां सन्धिवात हि ते मताः ॥
रसवातो रक्तवातो विषवातस्तथापरः । जीर्णवातो जरावातः पञ्च स्युस्तद्विदा इह॥ (Si.Ni.8/513-514)

In Sandhivāta, the Vāta gets aggravated and gradually afflicts the Sandhi (joints) and produces the Sandhi śūla (pain in the joints) and Sandhi śopha (swelling of the joints). Sandhivāta is classified into five types namely Rasavāta, Raktavāta, Viṣavāta, Jīrṇavāta and Jarāvāta.

- **Rasavāta:** The clinical features of Rasavāta are Ruja (pain) and Śopha (swelling) in Śākhā, Trika, Kaṭi, Pṛṣṭha, Grīva sandhīs, Sarvagātrapīḍa (pain all over the body) accompanied with Avipāka (indigestion), Aruci (anorexia) and Alpajvara vijvaroathavā (mild fever or no fever). It is also known as Āmavāta.
- **Raktavāta:** The clinical features of Raktavāta are Rugdāharāgayukta śotha (swelling associated with pain, burning sensation and redness), Sandhau ya śīghrasañcara (pain moves quickly from one joint to another) and Caraṇāṅguṣṭhamūlādau (especially affects the root of the big toe)
- **Viṣavāta:** The clinical features of Viṣavāta are Ruja (pain) and Śopha (swelling) in Jānvaṃsakaṭipṛṣṭhādau (knee, shoulder, waist, back etc.,) due to Phiraṅga and Pūyamehādi viṣa
- **Jīrṇavāta:** When the Sandhivāta become chronic and affects Nānāsandhisamāśraya (many joints) and Niścalīkurute sandhīn (makes them immovable), it is known as Jīrṇavāta.
- **Jarāvāta:** Jarāvāta is an Asthi vikṛti (bone disorder) mainly affecting the Pṛṣṭhavaṃśā (back and shoulder) and causes Kubjatā (state of being humpbacked) due to Jarāvastha (old age).

Sandhigatavāta **can be predominantly characterised by conditions like** Osteoarthritis (Please refer Topic 44)

KAṬIGRAHA

- वायुः कट्याश्रितः शुद्धः सामो वा जनयेद्रुजम् । कटिग्रहः....। (Ga.Ni.Dvi.19/160)

In Kaṭigraha, the aggravated Vāta either alone or along with Āma gets located in the Kaṭi (waist) producing Ruja (pain).

TRIKAŚŪLA

- ⦿ स्फिगस्थ्नोः पृष्ठवंशास्थ्नोर्यः सन्धिस्तत्त्रिकं मतम् । तत्र वातेन वा पीडा त्रिकशूलं तदुच्यते ॥ (B.P.M.24/114)

Sphigasthnoḥ pṛṣthavaṃśāsthnoryaḥ sandhi (the place where the hip bone joins with vertebral column) is known as Trika pradeśa. The aggravated Vāta afflicts the Trika pradeśa and produces the disease Trikaśūla.

Kaṭigraha and Trikaśūla can be predominantly characterised by conditions like low back ache, lumbar spondylolysis, sacroilitis, ankylosing spondylitis.

MANYĀSTAMBHA

- ⦿ दिवास्वप्नासनस्थानविवृताध्वनिरीक्षणैः । मन्यास्तम्भं प्रकुरुते स एव श्लेष्मणाऽऽवृतः ॥ (Su.Ni.1/67)

Due to Divāsvapna (sleeping during daytime), Āsana (Upaveśana - irregular sitting postures), Sthāna (Ūrdhvī bhavana - gazing upward) and Vivṛtādhvanirīkṣaṇai (Vakaramārgavalokanai - looking aside in twisted way), the Vāta gets aggravated and enveloped in the deranged Kapha and produces the disease Manyāstambha, which occurs as a premonitory symptom of Apatānaka.

Manyāstambha can be predominantly characterised by conditions like Torticollis, cervical degenerative disc disease and cervical osteoarthritis.

VĀTA KAṆṬAKA

- ⦿ न्यस्ते तु विषमं(मे) पादे रुजः कुर्यात् समीरणः । वातकण्टक इत्येष विज्ञेयः खडु(ल)काश्रितः ॥ (Su.Ni.1/79)

The Vāta located in the Khuḍaka (ankle joint) gets aggravated by Viṣamepāde (walking on the uneven roads) and produces Ruja (pain in the ankles). This disease is called Vāta Kaṇṭaka.

- ⦿ रुक् पादे विषमन्यस्ते श्रमाद्वा जायते यदा। वातेन गुल्फमाश्रित्य तमाहुर्वातकण्टकम्॥ (A.H.Ni.14/53)

The Vāta located in the Gulpha (ankle joint) gets aggravated by Viṣamepāde (walking on the uneven roads) or Pāde Śramāt (over exertion to the feet) and produces Ruja (pain in the ankles) and is called Vāta Kaṇṭaka

Vāta Kaṇṭaka can be predominantly characterised by conditions like calcaneal spur, **plantar** fasciitis

AṂSAŚOṢA

- ⦿ अंसदेशस्थितो वायुः शोषयित्वांऽसबन्धनम् । (Su.Ni.1/82)
- ⦿ अंसदेशस्थितो वायुः शोषयेदंसबन्धनम् ॥ (M.N.22/64)

The aggravated Vāta gets localized in the Aṃsadeśa and causes Aṃsabandha śoṣa (wasting of muscles in the shoulder joint)

- ⦿ अंसबन्धनं श्लेष्माणमित्यर्थः। अंसबन्धनं शोषयित्वा 'अंसशोषं रोगं करोति' इति । (Ḍa.Su.Ni.1/82)
- ⦿ अंसेत्यादिना श्लोकार्धेनांसशोषः केवलवातज उच्यते। अंसबन्धनकारकः श्लेष्मा अंसबन्धनः; एतदनन्तरं 'अंसशोषं जनयेत्' इति (Ma.M.N.22/64)
- ⦿ अंसबन्धनमिति अंसबन्धनकारकः श्लेष्मा अंसबन्धनः, तत्रांसबन्धनं श्लेष्माणं शोषयित्वा स तदनन्तरं अंसशोषं जनयेदिति वाक्यशेष इति (Āt.M.N.22/64)

Śleṣma (Kapha) is Aṃsabandhanakāraka, the binding element of the shoulder joint, and provides Aṃsabandhana (stability to the shoulder joint). When aggravated Vāta (Kevala Vāta - Vāta alone) dries up the Aṃsabandhana, it leads to the disease Aṃsaśoṣa.

AVABĀHUKA

- ⦿ अंसमूलस्थितो वायुः सिराः सङ्कोच्य तत्रगाः। बाहुप्रस्पन्दितहरं जनयत्यवबाहुकम्॥ (A.H.Ni.14/43)

In Avabāhuka, the aggravated Vāta gets localized in the Aṃsamūla and causes Sirā saṅkoca (constriction of blood vessels) resulting in Bāhupraspanditahara (loss of movement of the arm).

- ⦿ सिराश्चाकुञ्च्य तत्रस्थो जनयत्यवबाहुकम् ॥ (Su.Ni.1/82)
- ⦿ अवबाहुकः सन्धिगतेन कफस्नेहेन वातेन च, सन्धिगतत्वात्; अंससन्धेश्च कफस्थानत्वात्। (Ḍa.Su.Ni.1/82)

The aggravated Vāta causes Sirā saṅkoca (constriction of blood vessels) situated at Aṃsadeśa and produces the disease Avabāhuka.

Ḍalhaṇa, while commenting on the above verse, mentioned that the condition Avabāhuka arises from the combined influence of Kapha and Vāta in the Sandhi (joints), particularly manifesting in the Aṃsasandhi (shoulder joint), which is considered a site of Kapha.

Avabāhuka can be predominantly characterised by conditions like frozen shoulder, periarthritis, osteoarthritis of shoulder, shoulder impingement syndrome, bicipital tendonitis, and cervical myelopathy.

Asthimajjāgatavāta

- ⊙ भेदोऽस्थिपर्वणां सन्धिशूलं मांसबलक्षयः। अस्वप्नः सन्तता रुक् च मज्जास्थिकुपितेऽनिले॥ (Ca.Ci.28/33)

When vitiated Vāta is located in the Majjā (bone marrow) and Asthi (bones), it produces Bhedoasthiparvaṇāṃ (breaking type pain in the bones and joints), Sandhiśūla (pain in the joints), Māṃsabalakṣaya (loss of muscle and strength), Asvapna (loss of sleep) and Santata ruk (constant pain). Ācārya Suśruta added Ruja na praśāmyati (pain which does not subside) and Asthi śoṣa prabheda śūla (degeneration of bone with cracks and pain) as a symptom of Majjā (bone marrow) and Asthi (bones) respectively.

Difference between Avabāhuka and Viśvācī

Lakṣaṇās	Avabāhuka	Viśvācī
Saṃprāpti and Lakṣaṇa	The aggravated Vāta (Vātakapha) gets localized in the Aṃsamūla and causes Sirā saṅkoca resulting in Bāhupraspanditahara	The aggravated Vāta afflicts the Kaṇḍarā of the Tala, Pratyaṅgulīnāṃ and Bāhupṛṣṭhataḥ and produces Bāhuceṣṭāpaharaṇī or Bāhvoḥ karmakṣaya
Sthāna	Affects the shoulder joint and upper extremities	Affects the upper extremities
Modern parlance	Frozen shoulder, periarthritis, osteoarthritis of shoulder, shoulder impingement syndrome, bicipital tendonitis, and cervical myelopathy.	Cervical spondylolysis, Spondylolisthesis with radiculopathy, Brachial neuralgia, Thoracic outlet syndrome, Erb's paralysis

Short Answer questions

1. Describe Avabāhuka and Aṃsaśoṣa
2. Describe Asthimajjāgata Vāta
3. Write the differences between Avabāhuka and Viśvācī

Long Answer questions

1. Explain Nirūkti, Saṃprāpti, Pūrvarūpa, Bheda, and Lakṣanās of Sandhigatavāta

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TOPIC 44

Diseases of Bones, Joints and Muscles

Introduction

Disorders affecting the bones, joints, and muscles, collectively referred to as musculoskeletal disorders; exert a significant impact on the structural and functional integrity of the human body. This system encompasses bones, muscles, tendons, ligaments, joints, and cartilage. These conditions manifest through symptoms such as pain, stiffness, restricted mobility, and disability, thereby influencing daily activities and overall well-being. The common disorders include osteoarthritis, osteoporosis, frozen shoulder, calcaneal spur, plantar fasciitis, tennis elbow, carpal tunnel syndrome, and muscular dystrophy.

OSTEOARTHRITIS

Introduction

Osteoarthritis is the most common type of arthritis and is characterised by progressive disorder of the joints caused by gradual loss of cartilage and resulting in the development of bony spurs and cysts at the margins of the joints. It is also known as Osteoarthrosis, degenerative joint disease and wear and tear arthritis. Osteoarthritis is the second most common musculoskeletal condition with a prevalence of 22% to 39% in India. Osteoarthritis is more common in women than men and more prevalent with ageing and is the leading cause of pain and chronic disability among the elderly.

Classification:

Osteoarthritis is classified into two types based on the causative factors, namely primary osteoarthritis and secondary osteoarthritis.

- ⊙ **Primary osteoarthritis** is more common type of arthritis and the cause is unknown, hence it is also known as idiopathic osteoarthritis. This condition commonly affects hands, feet, hips and knees as localized primary osteoarthritis. If primary osteoarthritis involves multiple joints, then it is known as generalized primary osteoarthritis.
- ⊙ **Secondary osteoarthritis** is associated with some predisposing factor responsible for the degenerative changes; they are trauma, metabolic abnormalities, congenital abnormalities, calcium deposition diseases, endocrine and neuropathic diseases, other bone and joint diseases etc.

Pathophysiology:

The articular cartilage, responsible for covering the articulating surfaces of bones within a joint, undergoes degradation attributed to mechanical stress and biochemical changes. This degradation results in the erosion of the smooth articulating surface, leading to interfacial friction between bones. In response to the damage, Chondrocytes, the cells in cartilage, attempt to repair the damage by producing enzymes that degrade collagen and proteoglycans. However, this repair process is often insufficient and leads to further cartilage breakdown. The progressive cartilage deterioration prompts adaptive changes in the underlying bone, ultimately culminating in the formation of osteophytes. These osteophytes give rise to structural alterations within the joint, consequently contributing to sensory manifestations of pain and stiffness. Concurrently, the synovial lining becomes inflamed, initiating the release of pro-inflammatory cytokines and proteases, further exacerbating cartilage degeneration and inciting joint inflammation. The cumulative effects of cartilage loss and bony remodeling lead to the narrowing of the joint space, a characteristic feature evident through radiographic imaging and diagnostic of osteoarthritis.

Clinical features:

Pain in and around the affected joint is the cardinal symptom. Usually it begins gradually as intermittent, localized, deep ache, often aggravated by weight bearing and relieved by rest but can eventually become constant. Stiffness of the joints follows awakening or after rest but lasts for less than 30 minutes and lessens with movement. Examination of affected joint shows restricted movements, tenderness, warmness, bony crepitus (crackling or grinding noise with movement), bony or soft tissue swelling, gross deformity, muscle weakness and wasting and mild or absent synovitis.

Diagnosis:

Diagnosis is based on clinical features and imaging studies. X-ray shows subchondral bone sclerosis, narrowing of joint spaces, osteophyte formation, joint destruction, subchondral cyst, loose bodies, effusions and joint mal-alignment. In some cases, MRI and CT scan helps to *identify* the underlying causes of secondary osteoarthritis. Arthroscopy helps to *identify* the *early* fissuring and surface erosion of the cartilage.

OSTEOPOROSIS

Introduction

Osteoporosis is a systemic skeletal condition characterized by low bone mass and micro-architectural deterioration of bone tissue, resulting in increased bone fragility and increased risk of fractures, particularly in the femoral neck, vertebral bodies, and distal radius. Globally, the condition affects 18.3% of the population, with a higher prevalence in women (23.1%) than in men (11.7%). In India, approximately 61 million people have osteoporosis, with 80% of cases occurring in women.

Classification:

Osteoporosis is classified into two types based on the factors affecting the metabolism of bone, namely primary osteoporosis and secondary osteoporosis.

- ⊙ **Primary osteoporosis** occurs in both men and women without a specific cause. It is most common in postmenopausal women and older men. Factors such as gonadal insufficiency, low calcium and vitamin D levels, certain medications, and hyperparathyroidism can accelerate bone loss. Inadequate calcium intake during adolescence can also prevent achieving optimal peak bone mass.
- ⊙ **Secondary osteoporosis** stems from factors other than aging and menopause, such as pathological conditions like chronic kidney disease, secondary hyperparathyroidism, elevated serum phosphorus, calcitriol deficiency, abnormalities of serum calcium and vitamin D, osteomalacia, and medications such as chronic use of corticosteroids, anti-seizure drugs, heparin, ethanol, tobacco, proton pump inhibitors, and H2 blockers. These factors contribute to deprivation of bone mass and elevated fracture risk.

Pathophysiology:

Bone remodeling occurs as a continuous process characterized by the resorption of old bone tissue by osteoclasts and the formation of new bone by osteoblasts. Regulatory factors include parathyroid hormone (PTH), calcitonin, estrogen, vitamin D, various cytokines, and local factors such as prostaglandins. In the context of osteoporosis, this equilibrium is disrupted, leading to an increased rate of bone resorption relative to bone formation. The three principal mechanisms contributing to the development of osteoporosis are inadequate peak bone mass during growth, excessive bone resorption, and inadequate bone formation during remodeling, possibly due to a bias of mesenchymal stem cells away from osteoblasts and toward the marrow adipocyte lineage. The interplay of these mechanisms underlies the development of fragile bone tissue.

Clinical features:

Patients with osteoporosis are typically asymptomatic unless a fracture has occurred. Non-vertebral fractures generally present with symptoms, while approximately two-thirds of vertebral compression fractures are asymptomatic, although patients may experience underlying chronic back pain due to other conditions such as osteoarthritis. Symptomatic vertebral compression fractures typically manifest as acute-onset pain that does not typically radiate, is exacerbated by weight bearing, may be accompanied by localized spinal tenderness, and usually begins to subside within 1 week. Persistent pain may endure for months or become chronic, in which case additional fractures or underlying spine disorders, including malignancy or infection, should be considered.

Diagnosis:

The determination of diagnosis relies on the measurement of bone mineral density (BMD) through Dual-energy X-ray absorptiometry (DEXA).

A DEXA scan provides the t-score and z-score. The t-score represents the variance between the measured bone mineral density and the mean value of bone mineral density in young adults, calculated in standard deviations. The World Health Organization (WHO) has established the normal bone mineral density for women as a t-score within one standard deviation of the young adult mean. Scores ranging from -1 to -2.5 indicate a diagnosis of osteopenia, while scores below -2.5 signify a diagnosis of osteoporosis.

FROZEN SHOULDER

Introduction

Frozen Shoulder, also known as Adhesive capsulitis (AC), is an inflammatory condition characterized by persistent shoulder pain lasting more than three months. This condition gives rise to fibrosis of the glenohumeral joint capsule, resulting in gradual and significant stiffness and restricted range of motion, particularly in external rotation. While symptoms may manifest suddenly, the recovery phase is prolonged, often spanning 2 to 3 years. However, a majority of cases exhibit satisfactory recovery outcomes. In India, the prevalence of frozen shoulder is similar to global estimates, affecting about 2-5% of the population. Studies indicate that the condition is more prevalent among older adults and those with diabetes.

The causes of frozen shoulder are not completely understood; however, several potential risk factors have been identified. These include diabetes mellitus, hypothyroidism, lung carcinoma, myocardial infarction, cardiac surgery and shoulder trauma.

Pathophysiology:

The initial phase involves inflammation of the synovial lining of the shoulder joint capsule, resulting in pain and a progressive restriction of shoulder motion. Subsequently, the thickening and tightening of the shoulder capsule occur due to the formation of fibrous adhesions. This process is accompanied by a decrease in synovial fluid volume within the joint, which ordinarily serves to lubricate the shoulder and facilitate smooth movement.

Clinical Features:

The symptoms of a frozen shoulder include the abrupt onset of unilateral anterior shoulder pain. Typical symptoms include restrictions in passive and active range of motion, initially affecting external rotation and later abduction of the shoulder. Depending on the stage and severity, the condition is usually self-limiting but can interfere with daily living, work, and leisure activities. Another common condition associated with a frozen shoulder is neck pain, often caused by the overuse of cervical muscles to compensate for the loss of shoulder motion.

Diagnosis:

Diagnosis of frozen shoulder is typically based on a person's medical history, physical examination, and imaging tests. It is often a diagnosis of exclusion, as other potential causes of shoulder pain and stiffness must be ruled out first. During a physical exam, frozen shoulder may be diagnosed if the limits of active range of motion are similar to the limits of passive range of motion. The most severely affected movement is usually external rotation of the shoulder. Imaging studies are used to rule out other conditions rather than confirming the diagnosis of frozen shoulder.

CALCANEAL SPUR

Introduction

A calcaneal spur, more commonly referred to as a heel spur, is a bony outgrowth that develops on the calcaneal bone. These spurs may manifest either at the back of the heel (dorsal heel spur) or beneath the sole (plantar heel spur). Dorsal spurs are frequently linked to achilles tendinopathy, while those under the sole are associated with plantar fasciitis.

In India, approximately 11% of the population exhibits calcaneal spurs, with a study indicating that 59% of individuals experiencing heel pain have these spurs. Incidence rates are subject to variability based on factors such as age, gender, and lifestyle. Notably, calcaneal spurs are more prevalent in the 40-50 age groups and slightly more common in women.

Risk factors for calcaneal spurs include conditions that put more pressure on the heel, such as obesity, prolonged standing, working on rigid surfaces, overtraining, and flat feet.

Furthermore, individuals with bone and joint disorders such as osteoarthritis, rheumatoid arthritis, gout, and ankylosing spondylitis are also more likely to develop calcaneal spurs.

Pathophysiology:

The main cause of calcaneal spur is the repetitive stress and strain on the calcaneal bone and the attached soft tissues. This mechanical stress leads to inflammation of the plantar fascia, a thick band of tissue that runs along the bottom of the foot and attaches to the heel bone. This inflammation is known as plantar fasciitis. In response to the inflammation and stress, the body deposits calcium at the site of the inflammation, leading to the formation of a bony protrusion or spur.

Clinical features:

A calcaneal spur is often asymptomatic but can cause pain in the area surrounding the spur. This pain usually worsens in the morning upon waking up and after long periods of rest. It may also occur at the end of the day or after standing for extended periods of time. Furthermore, engaging in high-impact activities like running, jumping, or training barefoot can worsen the symptoms.

Diagnosis:

The diagnosis of calcaneal spur typically involves clinical assessment and X-rays. Clinical evaluation helps distinguish whether heel pain is due to heel spurs or another condition, such as plantar fasciitis, degeneration of the heel fat pad, or compression of the foot's nerve structures (such as entrapment of the inferior calcaneal nerve or Baxter neuropathy). On a lateral X-ray view of the foot, plantar heel spurs appear as small spike-like projections of the calcaneal tuberosity at the base of the heel. In contrast, dorsal heel spurs are located above the insertion of the Achilles tendon.

PLANTAR FASCIITIS

Introduction

Plantar fasciitis is an inflammatory condition affecting the plantar fascia of the foot. This ailment arises from degenerative irritation at the origin of the plantar fascia, situated at the medial calcaneal tuberosity of the heel and the adjoining perifascial structures. Notably, the plantar fascia plays a pivotal role in foot biomechanics by providing arch support and absorbing shock during walking and running. It stands as the predominant cause of infra calcaneal pain, encompassing 80% of reported heel pain cases. Epidemiologically, it exhibits an estimated prevalence of 10% and is most frequently observed within the age range of 40 o 60 years.

Plantar fasciitis is often caused by overuse, resulting in micro-tears of the plantar fascia. Additionally, this condition may occur due to trauma or other multifactorial causes. Some contributing factors include pes planus (flat feet), pes cavus (high-arched feet), restricted ankle dorsiflexion, inappropriate footwear, and prolonged standing or jumping. Other risk factors include obesity, heel pad atrophy, ageing, occupations that involve prolonged standing, and weight-bearing. Although plantar fasciitis is associated with various seronegative spondyloarthropathies, systemic factors are unknown in approximately 85% of cases.

Pathophysiology:

Prolonged stress and strain on the plantar fascia can result in the formation of micro-tears within the tissue, leading to its degeneration over time. This degenerative process often denoted as "fasciosis" or "fasciopathy," primarily manifests as collagen degeneration at the plantar fascia's origin, particularly at the calcaneal tuberosity. While the principal pathology is degenerative, an inflammatory response may ensue as a secondary consequence of the micro-tears and degeneration, contributing to the pain and discomfort experienced by afflicted individuals.

Clinical Features:

Patients commonly present with a history of progressive pain localized at the inferior and medial heel. In severe cases, the pain may radiate proximally. The pain is typically described as sharp and is most pronounced during the initial steps in the morning. Prolonged periods of standing, and in severe instances, extended sitting, exacerbate the symptoms. Pain diminishes with ambulation or the commencement of physical activity but progressively intensifies throughout the day with increased activity. Reproduction of the pain can be achieved through palpation of the plantar medial calcaneal tubercle at the site of the plantar fascial insertion on the heel bone.

Furthermore, passive dorsiflexion of the foot and toes can elicit the pain. Acute, severe heel pain, especially when accompanied by mild local swelling, may signify an acute fascial tear. Additionally, some patients may report experiencing a burning or sticking pain along the plantar medial border of the foot during ambulation.

Diagnosis:

Plantar fasciitis is usually diagnosed through clinical evaluation. Additional tests are only needed if the diagnosis is uncertain. X-rays can be used to rule out any bone damage, especially if there is a history of injury, and to check for a plantar heel spur, which can indicate abnormal stress on the plantar fascia. An MRI scan may be recommended if there is ongoing uncertainty about the diagnosis, as it can reveal areas of thickening in the plantar fascia and any associated swelling.

TENNIS ELBOW

Introduction

Tennis elbow, also known as lateral epicondylitis, is characterized by the micro-tearing of fibers in the common extensor tendon at the origin of the extensor carpi radialis brevis (ECRB) tendon. Contrary to its name, only 5% of individuals with tennis elbow develop the condition from playing tennis. The primary etiology is repetitive stress on the tendon near the humerus, commonly stemming from activities such as extensive computer use, heavy lifting, forceful forearm movements, and repetitive vibration. Additionally, it is prevalent in other sports including squash, badminton, baseball, swimming, and field throwing events. Occupations involving repetitive one-sided movements, such as those of electricians, carpenters, gardeners, and sedentary workers, also predispose individuals to this condition.

The estimated prevalence of tennis elbow in the general populace ranges from 1% to 3%. It is particularly pervasive in individuals aged 40 to 50 years, with an approximate 10% prevalence among women in this age group. Risk factors for developing this condition in the broader population encompass smoking, obesity, engaging in repetitive movements for at least two hours daily, and involvement in strenuous activities requiring the management of physical loads exceeding 20 kg.

Pathophysiology:

Microtearing of the extensor carpi radialis brevis (ECRB) tendon is often caused by repetitive wrist extension and forearm pronation, leading to tendinosis. This degenerative process involves disorganization of collagen fibers and angiofibroblastic hyperplasia, resulting in weaker, disorganized tissue. Recent studies suggest reduced inflammation's role, with the condition primarily involving tendon degeneration rather than acute inflammation. The damaged tendons and surrounding tissues cause pain, especially during gripping or wrist extension activities.

Clinical Features

The primary symptom is pain localized to the outer aspect of the elbow, with potential radiation proximally along the upper arm and distally along the lateral aspect of the forearm, and rarely to the third and fourth digits. Aggravation of this pain is commonly associated with activities involving wrist extension, such as lifting, gripping, or rotational movements (e.g., turning a doorknob or opening a jar). Furthermore, tenderness over the lateral epicondyle, diminished grip strength, elbow stiffness, and occasional edema and a burning sensation in the affected area may be present.

Diagnosis

Diagnosis is based on physical examination and imaging studies. On examination, a positive **Cozen's test** is indicated by pain at the lateral epicondyle when the wrist is extended against resistance. The **chair pick-up test** is positive if the patient experiences pain at the lateral epicondyle while attempting to lift a chair using a three-finger pinch (thumb, index finger, and middle finger). **Mill's test** is positive if the patient experiences pain at the lateral epicondyle when the wrist is passively stretched in flexion and pronation while the elbow is extended. The **coffee cup test** is positive if the patient experiences pain when grasping a cup or any other object.

Imaging studies, such as elbow X-rays, reveal calcification at the extensor muscle attach to the lateral epicondyle. Ultrasonography and MRI are used to evaluate tears, stress fractures, or osteochondral defects. Electromyography (EMG) may be utilized to rule out nerve compression.

CARPEL TUNNEL SYNDROME

Introduction

Carpal tunnel syndrome (CTS), also known as nerve compression syndrome, is characterized by the compression of the median nerve at the carpal tunnel in the wrist. The primary contributing factor to the onset of CTS is the elevated pressure within the carpal tunnel. The typical initial manifestations of CTS include pain, numbness, and paresthesias, which affect the first three digits and the lateral half of the fourth digit.

The incidence of CTS in the general population ranges from 1% to 5%. CTS is more prevalent in females than males, with a 3:1 female-to-male ratio and typically manifests in adults aged 40 to 60. The risk of developing CTS is doubled in individuals who have dislocation or subluxation of the carpus, fractures of the distal radius, wrist arthrosis, inflammatory arthritis, and infectious arthritis, acromegaly, cysts or tumors within the tunnel, pregnancy, menopause, obesity, kidney failure, hypothyroidism, use of oral contraceptives, congestive heart failure, diabetes, alcoholism, vitamin deficiency or toxicity, and exposure to toxins

Pathophysiology:

The carpal tunnel is a narrow passageway in the wrist, formed by the transverse carpal ligament at its upper boundary and the carpal bones at its lower boundary. Within this confined space, nine flexor tendons and the median nerve pass through. Constriction of the carpal tunnel, caused by conditions such as tenosynovitis, rheumatoid arthritis, or fluid retention (often seen in pregnancy), leads to swelling and increased pressure on the median nerve. This increased pressure can impede venous outflow, resulting in localized swelling. As a result, compression of the median nerve intensifies, compromising blood flow and leading to ischemia. The resulting nerve dysfunction causes sensory and motor deficits, characterized by numbness, tingling, pain, and weakness in the hand.

Clinical Features:

The characteristic symptoms of carpal tunnel syndrome encompass numbness, tingling or burning sensations localized in the ring finger's thumb, index, middle, and radial half. These symptoms predominantly manifest during the night and in the morning. They typically affect bilaterally but not necessarily at the same time.

Diagnosis:

Diagnosis of carpal tunnel syndrome relies on clinical examination and electrodiagnostic testing. A positive **Phalen's maneuver** is indicated by the patient experiencing tingling, numbness, or pain in the fingers innervated by the median nerve (typically the thumb, index, middle, and part of the ring finger) within 30 to 60 seconds when the wrists are forcefully flexed by pressing the dorsal surfaces of both hands together. A positive **Tinel's sign** is identified by a tingling sensation or a mild electric shock-like feeling in the nerve distribution area when the median nerve is lightly tapped just proximal to the flexor retinaculum. **Durkan's test** (Carpal tunnel compression test) is positive when the patient experiences paresthesia with firm pressure applied to the carpal tunnel over the nerve for up to 30 seconds. The **hand elevation test** is positive when paresthesia occurs in the median nerve distribution within 2 minutes of lifting both hands above the head.

Electrodiagnostic testing, such as Nerve conduction studies (Electroneurography (ENG)), may reveal diminished sensory and motor signals through the median nerve, particularly across the carpal tunnel. Electromyography (EMG) may demonstrate abnormal electrical activity in the muscles innervated by the median nerve, particularly during rest.

MUSCULAR DISEASES - MUSCULAR DYSTROPHY

Introduction

Muscular diseases encompass a wide range of conditions that primarily affect the human muscle system, resulting in skeletal muscle weakness. Primary muscle diseases arise from inherent abnormalities within the muscles themselves, such as polymyositis, dermatomyositis, muscular dystrophy, myasthenia gravis, amyotrophic lateral sclerosis (ALS), rhabdomyolysis, cardiomyopathy, and sarcopenia. Conversely, secondary muscle diseases stem from other underlying conditions that may have triggered or caused the muscle disease. In this context, the focus will be on the discussion of muscular dystrophy.

Muscular dystrophy encompasses a spectrum of genetic disorders characterized by progressive muscle weakness and atrophy. It affects an estimated 1 in 5,000 individuals globally and can present at any stage of life, with childhood being the most prevalent period for diagnosis. The condition arises from mutations impacting genes responsible for muscle structure and function, leading to gradual degeneration and loss of muscle fibers. Additionally referred to as myodystrophy and myodystrophia, the common types of muscular dystrophy, arising from X-linked and autosomal inheritance, include Duchenne, Becker, facioscapulohumeral, limb-girdle, and myotonic forms. Among these, Duchenne muscular dystrophy stands as the most prevalent manifestation of this condition.

- **Duchenne muscular dystrophy** (pseudohypertrophic muscular dystrophy or Xp21 myopathy) is X-linked recessive disorder, which occurs mainly in boys before the age of 5 years. In this type, the gene dystrophin, which is located in the short arm of the X- chromosome 21, is absent or grossly deficient. Duchenne muscular dystrophy is characterised by weakness of proximal and pelvic girdle muscles leading to repeated falls and weakness while climbing stairs and getting up from the ground. It is associated with mental impairment, cardiomyopathy and respiratory failure. The life span rarely exceeds 20 years. Clinically pseudohypertrophy of calf muscles and positive gower's sign (on getting up from the ground, the boy uses his hands to straighten up) are observed.
- **Becker muscular dystrophy** is also an X-linked recessive disorder, which occurs mainly in boys after the age of 5 years. The genetic defect is in the same place of gene dystrophin, but small amount of dystrophin can be synthesised and utilised by muscles. Hence, weakness of proximal and pelvic girdle muscles occurs slowly and less severe than DMD. In this type, the patient may be able to walk up to adolescence and live up to 40 years.
- **Facioscapulohumeral dystrophy** is an autosomal dominant disorder, which occurs in both sex but males are prominent and its onset may occur at any age from childhood to adult life. The genetic defect is an abnormal deletion of tandem 4q telomere gene and characterised by weakness in the face and shoulder girdle muscles leading to difficulty in whistling, difficulty in closing the eyes and difficulty in raising arms above the shoulder level associated with winging of scapula. Sensory neural deafness and coat's disease are seen in some cases and cardio- respiratory involvement is rare. Life expectancy is normal as the disease runs an insidious course. Due to biceps and triceps severely affected the forearm muscles looks hypertrophied, distal muscles of the lower limbs are affected asymmetrically and positive beevor's sign (the upward movement of the umbilicus towards the head on flexing the neck) are observed in majority cases.
- **Limb-Girdle muscular dystrophy** is most often inherited as autosomal recessive and less commonly inherited as autosomal dominant disorder, occurs in both sexes and its onset may occur at their second and fourth decades of life. There are at least 31 different sub types (23 autosomal recessive – LGMD R1 – R23 and 8 autosomal dominant – LGMD D1 – D8) of limb-girdle muscular dystrophy have been described

and it is characterised by progressive, symmetric and proximal muscle weakness in the limb girdle and shoulder girdle leading to difficulty in walking up stairs and difficulty in raising arms respectively with or without facial involvement. Cardiomyopathy and respiratory failure may develop in chronic disease. Many patients manage to live to late adulthood.

- **Myotonic dystrophy (Dystrophia Myotonica)** is an autosomal dominant disorder, which occurs in both sexes and its onset usually occurs between 20-30 years of age. This condition is classified in to two types namely Myotonic dystrophy type 1 (DM1) and Myotonic dystrophy type 2 (DM2) or Proximal myotonic myopathy (PROMM). In DM1, the genetic defect is unstable expansion of a CTG trinucleotide repeat in a serine-threonine protein kinase gene on chromosome 19q and in DM2, the genetic defect is mutation of the ZNF9 gene located on chromosome 3q. It is characterised by weakness of temporalis, masseter, sternocleidomastoids and facial muscles leading to hatchet faced appearance (thin, hard and unpleasant face) associated with ptosis and frontal baldness. Other features include Grip myotonia, cognitive impairment, early cataracts, day time sleepiness, hypogonadism, cardiomyopathy, respiratory insufficiency and endocrine dysfunction including increased risk of diabetes. Usually the disease lasts up to 50 years of age but premature death occurs due to cardiac arrhythmia or respiratory failure.

Diagnosis:

The diagnosis is confirmed through specific molecular genetic testing, electromyography (EMG) and characteristic muscle biopsy. Serum creatine kinase levels are significantly elevated in Duchenne and Becker muscular dystrophy, moderately elevated in facioscapulohumeral and limb-girdle muscular dystrophy, and within the normal range in myotonic dystrophy. It is essential to screen for associated cardiac and respiratory abnormalities. Genetic testing reveals deletions or duplications of the dystrophin gene in 65% of patients. Electromyography (EMG) demonstrates characteristic features of myopathy, while muscle biopsy reveals varying muscle fiber sizes and small groups of necrotic and regenerating fibers.

MCQ's

1. Which of the following is the most common type of arthritis

   a) Rheumatoid arthritis

   b) Osteoarthritis

   c) Psoriatic arthritis

   d) Gout

2. What is the primary characteristic of osteoarthritis

   a) Inflammation of the synovial membrane

   b) Progressive loss of cartilage in joints

   c) Autoimmune attack on joint tissues

   d) Deposition of uric acid crystals in joints

3. Which of the following is NOT a common site affected by primary osteoarthritis

   a) Hands b) Feet

   c) Elbows d) Knees

4. Which diagnostic tool is primarily used to measure bone mineral density (BMD) in osteoporosis

   a) MRI

   b) CT scan

   c) Dual-energy X-ray absorptiometry (DEXA)

   d) Ultrasound

5. What is another name for frozen shoulder

   a) Rotator cuff tear b) Adhesive capsulitis

   c) Shoulder impingement d) Glenohumeral arthritis

6. What characterizes frozen shoulder

   a) Persistent shoulder pain lasting more than three months

   b) Sudden onset of bilateral shoulder pain

   c) Increased range of motion in the shoulder

   d) Rapid recovery within a few weeks

7. Which of the following is a common risk factor for developing frozen shoulder

   a) Hyperthyroidism b) Diabetes mellitus

   c) Osteoporosis d) Rheumatoid arthritis

8. Which movement is most commonly restricted in frozen shoulder

   a) Internal rotation b) External rotation

   c) Flexion d) Extension

9. Which condition is commonly associated with dorsal heel spurs
a) Plantar fasciitis
b) Achilles tendinopathy
c) Osteoarthritis
d) Rheumatoid arthritis

10. Which of the following is NOT a contributing factor to plantar fasciitis
a) Pes planus (flat feet)
b) Pes cavus (high-arched feet)
c) Restricted ankle dorsiflexion
d) High calcium intake

11. What is another name for tennis elbow
a) Medial epicondylitis
b) Lateral epicondylitis
c) Rotator cuff tendinitis
d) Carpal tunnel syndrome

12. Which tendon is primarily affected in tennis elbow
a) Flexor carpi radialis
b) Extensor carpi radialis brevis
c) Biceps brachii
d) Triceps brachii

13. Which test is positive if the patient experiences pain at the lateral epicondyle when the wrist is extended against resistance
a) Mill's test
b) Cozen's test
c) Chair pick-up test
d) Coffee cup test

14. What is another name for carpal tunnel syndrome
a) Medial epicondylitis
b) Lateral epicondylitis
c) Nerve compression syndrome
d) Rotator cuff tendinitis

15. Which nerve is compressed in carpal tunnel syndrome
a) Ulnar nerve
b) Radial nerve
c) Median nerve
d) Sciatic nerve

16. Which test is positive if the patient experiences tingling, numbness, or pain in the fingers when the wrists are forcefully flexed by pressing the dorsal surfaces of both hands together
a) Tinel's sign
b) Phalen's maneuver
c) Durkan's test
d) Hand elevation test

17. Which type of muscular dystrophy is the most prevalent
a) Becker muscular dystrophy
b) Facioscapulohumeral dystrophy
c) Limb-Girdle muscular dystrophy
d) Duchenne muscular dystrophy

18. What is the genetic inheritance pattern of Duchenne muscular dystrophy
a) Autosomal dominant
b) Autosomal recessive
c) X-linked recessive
d) Mitochondrial inheritance

19. Which gene is affected in Duchenne muscular dystrophy
a) Dystrophin
b) Myosin
c) Actin
d) Troponin

20. Which type of muscular dystrophy is characterized by difficulty in whistling and closing the eyes
a) Duchenne muscular dystrophy
b) Becker muscular dystrophy
c) Facioscapulohumeral dystrophy
d) Limb-Girdle muscular dystrophy

21. Which type of muscular dystrophy is most often inherited as an autosomal recessive disorder?
a) Duchenne muscular dystrophy
b) Becker muscular dystrophy
c) Facioscapulohumeral dystrophy
d) Limb-Girdle muscular dystrophy

Answer Keys

1	b	2	b	3	c	4	c	5	b	6	a	7	b	8	b
9	b	10	d	11	b	12	b	13	b	14	c	15	c	16	b
17	d	18	c	19	a	20	c	21	d						

Short Answer Questions

1. Explain Osteoarthritis
2. Explain Osteoporosis
3. Write the difference between primary and secondary osteoarthritis
4. Differentiate Osteoarthritis and Osteoporosis
5. Explain Frozen shoulder
6. Explain Calcaneal spur
7. Explain Plantar fasciitis
8. Differentiate Calcaneal spur and Plantar fasciitis
9. Explain Tennis elbow
10. Explain Carpel tunnel syndrome
11. Explain muscular dystrophy

TOPIC 45

Āmavāta

Introduction

Āmavāta is one of the diseases of Rasavaha srotas and is the result of Agniduṣṭi (impaired Agni), Āmotpatti (formation of Āma) and Sandhivikṛti (diseases of the joints). The description of Āmavāta is not available in Bṛhatrayīs like Caraka Saṃhitā, Suśruta Saṃhitā, and Aṣṭāṅga Hṛdaya. Ācārya Mādhavakara is the first one to describe this condition as a separate disease in Mādhava Nidāna. The term Āmavāta is formed by the union of two words Āma and Vāta; and these two play a crucial role in the pathogenesis of this disease. In Siddhānta Nidānam of Kaviraj Gananāth Sen, the word Rasavāta has been described as a synonym of the disease Āmavāta.

Nirūkti and Paribhāṣa

- आमोऽपाकहेतुः वातः। स्वनामख्यातरोगविशेषः॥ (SKD)

Āma produced due to indigestion along with Vāta causes Āmavāta. It is a disease that can be known by its name itself.

- आमेन दुष्टो वात आमवातः॥ (Āḍ.Śā.P.7/41)

Due to Āma, Vāta aggravates and produces the disease 'Āmavāta'.

- आमेन युक्तो वात आमवातः स तु शोथादियुक्तस्त्रिकसन्धिवेदनावान्॥ (Kā.Śā.P.7/41)

Āmavāta is a condition where clinical features like Śotha (swelling) etc., appear over the Trikāsandhi along with Vedanā (pain) due to the vitiation of Āma and Vāta.

- युगपत्कुपितावन्तस्त्रिकसन्धिप्रवेशकौ । स्तब्धं च कुरुतो गात्रमामवातः स उच्यते ॥ (M.N.25/5)

The aggravated doṣas (Āma and Vāta) enters together into the Trikāsandhi (sacral joints) and produces Gātra stabdha (stiffness of the body) is called as Āmavāta.

Nidāna of Āmavāta

- विरुद्धाहारचेष्टस्य मन्दाग्नेर्निश्चलस्य च । स्निग्धं भुक्तवतो ह्यन्नं व्यायामं कुर्वतस्तथा ॥ (M.N.25/1)

Indulgence in Viruddhāhāraceṣṭa (incompatible food and regimen), Mandāgni (subdued digestive fire), Niścālasya (lack of physical activity), Snigdhaṃ bhuktavato hyānnaṃ vyāyāmaṃ (doing exercise after eating unctuous food) are the causative factors of Āmavāta.

Samprāpti of Āmavāta

- वायुना प्रेरितो ह्यामः श्लेष्मस्थानं प्रधावति । तेनात्यर्थं विदग्धोऽसौ धमनीः प्रतिपद्यते ॥
  वातपित्तकफैर्भूयो दूषितः सोऽन्नजो रसः । स्रोतांस्यभिष्यन्दयति नानावर्णोऽतिपिच्छिलः ॥
  जनयत्याशु दौर्बल्यं गौरवं हृदयस्य च । व्याधीनामाश्रयो ह्येष आमसञ्ज्ञोऽतिदारुणः ॥ (M.N.25/2-4)

Due to indulgence in above said causative factors, Āma is formed in the body. This Āma being propelled by the vitiated Vāta gets lodged in the Śleṣma sthāna (seats of Kapha, i.e. Āmāśaya, Sandhi and Hṛdaya) of the body, then being excessively Vidagdha (improperly digested) it circulates through the Dhamanīs (vessels). This Anna rasa further vitiated by Vāta, Pitta and Kapha doṣa causes Srotāṃsya abhiṣyanda (obstruction to the channels of circulation) and is Nānāvarṇā (different colours) and Atipicchila (extremely slimy). Consequently it causes Agni daurbalya (suppresses the digestive fire) and Hṛdayasya gauravaṃ (heaviness in the heart). And this Āma becomes Vyādhīnamaśraya (root cause of many diseases) and is Atidāruṇaḥ (very dreadful).

- जङ्गरानलदौर्बल्यादविपक्वस्तु यो रसः। स आमसंज्ञको देहे सर्वदोषप्रकोपनः॥ (Y.R.28/9)

Due to Agni daurbalya (depletion of digestive fire), the ingested Anna rasa remains Vipakva (undigested) and this is called Āma. This Āma causes Sarvadoṣaprakopa (vitiation of all the doṣas of the body).

- अजीर्णोन्नरसो जातः क्रमशः सञ्जितश्च यः। आमसंज्ञां स लभते शिरोगात्ररुजाकरः॥ (Y.R.28/5)

The undigested Anna rasa of the ingested food gets gradually accumulated. This is called Āma and it causes Śirogātra rujā (pain in the head and whole body).

Samprāpti Ghaṭaka

Doṣa	Vāta Pradhāna Tridoṣa
Dūṣya	Rasa, Māṃsa, Asthi, Majjā, Sandhi, Snāyu
Agni	Jaṭharāgni, Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhya janya
Srotas	Anna, Rasa, Māṃsa, Asthi, Majjā vaha

Srotoduṣṭi	Saṅga
Udbhava sthāna	Āmāśaya
Sañcāra sthāna	Sarva rasavāhini
Adhiṣṭhāna	Śleṣma sthāna especially Sandhi
Rogamārga	Madhyama
Vyādhi svabhāva	Cirakāri

Pūrvarūpa of Āmavāta

- अजीर्णाद्यो रसो जात: संचित: संक्रमेण वै। आमसंज्ञां स लभते शिरोगात्ररुजाकर:॥ (Va.Sa.31/5)

When the food taken is not digested properly, the ingested food accumulates and produces the condition Āma. This Āma causes Śirorujā (headache) and Gātrarujā (body ache).

Bheda

Āmavāta is classified into two types based on the Avasthā (different stage) of the disease namely Sāmānya avasthā and Pravṛddha avasthā

- एकदोषानुगः साध्यो, द्विदोषो याप्य उच्यते । सर्वदेहचरः शोथः स कृच्छ्रः सान्निपातिकः ॥ (M.N.25/12)

Ācārya Mādhavakara and Yogaratnākara classified Āmavāta into seven types based on the involvement of doṣas namely Vātaja, Pittaja, Kaphaja, Vātapittaja, Pittakaphaja, Kaphavātaja and Sannipātaja.

- आमं संक्षयते प्राज्ञश्चतुर्धा भेदलक्षणै:॥विष्टम्भी गुल्मकृन्स्नेही आम: पक्वाम एव च।
  सर्वांगो भवेच्चान्यो वक्ष्ये तस्यापि लक्षणम्॥ (Hā.Sa.21/5-6)

Ācārya Hārīta classified Āmavāta into four types based on the Lakṣaṇās (symptoms) namely Viṣṭambhī āma, Gulmakṛta āma, Snehī āma and Pākvāma. Sarvāṅga āma is also considered as another type of Āmavāta.

Based on Kāla (time period), Āmavāta can be classified into 2 types based namely Navīna (less than one year) and Purāṇa (more than one year).

Sāmānya Lakṣaṇās of Āmavāta

- अङ्गमर्दोऽरुचिस्तृष्णा आलस्यं गौरवं ज्वरः । अपाकः शूनताऽङ्गानामामवातस्य लक्षणम् ॥ (M.N.25/6)

Aṅgamarda (body ache), Aruci (anorexia), Tṛṣṇā (thirst), Ālasya (laziness), Gaurava (heaviness), Jvara (fever), Apāka (indigestion) and Aṅga śūnam (swelling of the body parts) are the clinical features of Āmavāta.

- मन्यापृष्ठकटीजानुत्रिकसन्धीन्विकुञ्चयन्। सशब्दः स्रस्तगात्रश्च आमवात: स उच्यते॥ (Y.R.28/8)

Vikuñjana (contracture) of Manyā (neck), Pṛṣṭa (back), Kaṭī (waist), Jānu (knee) and Trikā sandhi (sacral joint), along with Śabda (makes sound while moving those joints) and Srastagātra (slackened body parts) are the features of Āmavāta.

- शाखा त्रिक कटी पृष्ठ ग्रीवादिषु हि सन्धिषु। क्वचिच्छोफरुजाकारी सर्वगात्रप्रपीडन:॥
  अविपाकाऽरुचियुतोऽल्पज्वरो विज्वरोऽथवा। रसवातगद: सोऽयमामवातश्च कथ्यते॥ (Si.Ni.8/515-516)

Śopha (swelling) and Ruja (pain) in the Śākhā sandhis (joints of the extremities), Trika (sacrum), Kaṭī (waist), Pṛṣṭa (back), Grīvā (neck) etc., Sarvagātra Piḍana (squeezing type of pain in the whole body) associated with Avipāka (indigestion), Aruci (anorexia), Alpajvara (mild fever) or Vijvara (no fever) are the features of Rasavāta; and it is also called as Āmavāta.

Pravṛddha Āmavāta Lakṣaṇa

- स कष्टः सर्वरोगाणां यदा प्रकुपितो भवेत् । हस्तपादशिरोगुल्फत्रिकजानूरुसन्धिषु ॥
  करोति सरुजं शोथं यत्र दोषः प्रपद्यते । स देशो रुज्यतेऽत्यर्थं व्याविद्ध इव वृश्चिकैः ॥
  जनयेत् सोऽग्निदौर्बल्यं प्रसेकारुचिगौरवम् । उत्साहहानिं वैरस्यं दाहं च बहुमूत्रताम् ॥
  कुक्षौ कठिनतां शूलं तथा निद्राविपर्ययम् । तृट्छर्दिभ्रममूर्छाश्च हृद्ग्रहं विड्विबद्धताम् ।
  जाड्यान्त्रकूजमानाहं कष्टांश्चान्यानुपद्रवान् ॥ (M.N.25/7-10)

When this Āma is vitiated, it becomes the most difficult among all diseases and causes Saruja śotha (pain along with swelling) in the Sandhi (joints) of Hasta (arms), Pāda (feet), Śiras (head), Gulpha (ankle joint), Trika sandhi (sacral joint), Jānu (knee joint) and Ūru (thighs). Wherever it reaches, it produces Vṛścika vyāviddha iva atyartha ruja (severe pain as though bitten by a scorpion). Agni daurbalya (poor digestion), Praseka (salivation), Aruci (anorexia), Gaurava (heaviness), Utsāha hāni (lack of enthusiasm), Vairasya (bad taste in the mouth), Dāha (burning sensation), Bahu mūtrata (excessive urination), Grahaṇī doṣa (sprue), Kukṣau kaṭhinatāṃ śūlaṃ (hardness and pain of the abdomen), Nidrāviparyaya (loss of sleep), Tṛṭ (thirst), Chardi (vomiting), Bhrama (giddiness), Mūrcchā (fainting), Hṛdgraha (pain in the heart), Viḍvibandham (constipation), Jāḍyā (rigidity of the body), Āntrakūjanam (gurgling sounds in the intestines), Ānāha (abdominal distention with constipation) and Anyānupadravā (many such complications) develops.

Doṣaja Āmavāta Lakṣaṇās

- पित्तात्सदाहरागं च सशूलं पवनानुगम्। स्तैमित्यं गुरुकण्डूकं कफजुष्टं तमादिशेत्॥ (Y.R.28/15)

In case of Pitta predominance, there is Dāha (burning sensation) and Rāga (redness), in Vāta predominance, there is Śūla (pain) and in Kapha predominance, there is Staimityaṃ (as if body is covered with moisture), Guru (heaviness) and Kaṇḍū (itching sensation).

Lakṣaṇa bheda Lakṣaṇās

Viṣṭambhī Āmavāta

- ⦿ विष्टम्भी गुरुचाध्मानं बस्तिशूलं च जायते॥ (Hā.Sa.21/7)

Viṣṭambhī (constipation), Guru (heaviness), Ādhmāna (abdominal distention) and Basti Śūla (pain in the urinary bladder) are the features of Viṣṭambhī Āmavāta.

Gulmī Āmavāta

- ⦿ जङ्गरं गर्जते यस्य गुल्मवत्परिपीड्यते। कटिदेशे जडत्वञ्च आमगुल्माभिशङ्कित:॥ (Hā.Sa.21/8)

Jaṭhara garjate (gurgling sound in the intestines), Gulmavatparipīḍayate (severe pain like Gulma) and Kaṭideśa jaḍatvam (stiffness in the waist) are the features of Gulmī Āmavāta.

Snehī Āmavāta

- ⦿ यस्य च स्निग्धता गात्रे जाड्यं मन्दाग्निको बली। स्नेहामो विजलो यस्य स्नेहो वाम: प्रकीर्त्तित:। (Hā.Sa.21/10-11)

Gātra Snigdhatā (unctuousness of the body), Jāḍyaṃ (spasticity), Mandāgni (subdued digestive fire) and Snehāmo vijalo (excretion of unctuous āma devoid of water) are the features of Snehī Āmavāta.

Pakvāmavāta

- ⦿ सपीतो विजल: श्यामा: पक्वाम: पतते त्वध:। न बस्तिशूलो भवति आमवाते श्रम: क्लम:॥ (Hā.Sa.21/15)

Pīta vijala śyāmā pakvāmaḥ patate tvadhaḥ (excretion of yellowish black pakva āma devoid of water) along with Śrama (tiredness), Klama (exhaustion) and Na basti Śūla (absence of pain in the urinary bladder).

Sarvāṅga Āmavāta

- ⦿ कटिपृष्ठे वक्षोदेशे तोदनं वस्तिशूलवान्। गुल्मतो जङ्गरं गर्जेत्तथात: शोफ एव च॥
  शिरोगुरुत्वं भवति आमश्च पतते भृशम्। सर्वांगो भवेत्सोऽपि विज्ञेयोऽसौ विजानत॥ (Hā.Sa.21/17)

Kaṭipṛṣṭhe vakṣodeśe todanaṃ (pricking pain in the waist, back and chest), Basti śūla (pain in the urinary bladder), Gulmato jaṭharaṃ garjate (intestinal gurgling sounds resembling Gulma), Śopha (swelling), Śirogurutva (heaviness in the head) and Āmaśca patate bhṛṣam (excreting Āma in excess) are the features of Sarvāṅga Āmavāta.

Upadravā of Āmavāta

- ⦿ अन्यानुपद्रवान् सङ्कोचखञ्जत्वादीन्॥ (Ma.M.N.25/7-10)

Ācārya Vijayarakṣita stated Saṅkoca (contraction of the body parts) and Khañjatvā (lameness) etc., as the complications of Āmavāta.

- ⦿ जाड्यान्त्रकूजनानाहतृट्छर्दिबहुमूत्रता। शूलं शयननाशोऽष्टोपद्रवा आमवातजा:॥ (Añ.Ni.1/119)

Jāḍya (spasticity), Āntrakūjana (gurgling sound in the intestines), Ānāha (abdominal distention with constipation), Tṛṭ (thirst), Chardi (vomiting), Bahumūtrata (excessive urination), Śūla (pain) and Śayana nāśa (loss of sleep) are the eight complications of Āmavāta.

Sādhyāsādhyata

- ⦿ एकदोषानुगः साध्यो, द्विदोषो याप्य उच्यते । सर्वदेहचरः शोथः स कृच्छ्रः सान्निपातिकः ॥ (M.N.25/12) / (Y.R.28/16)

The disease is easily curable if only one doṣa is involved; difficult to cure if two doṣas are involved and it becomes incurable if all the three doṣas are involved and also in the presence of Sarvadehacara śotha (generalized swelling of the body).

In Contemporary science,

In the case of Āmavāta, there is difference of opinion between the Keraliya Āyurveda tradition and the Rest of Indian Āyurveda practitioners. This is because of their differences in understanding the disease viz., Keraliya tradition considered Āmavāta as Rheumatic fever with its complications or early / acute phase of Vātarakta (Āmāvastha of Vātarakta) whereas others considered it as Rheumatoid arthritis.

Āmavāta can be correlated to certain stages of Rheumatoid arthritis, Ankylosing spondylitis, Rheumatic fever, Systemic lupus erythematosus etc.

Short Answer Questions

1. Explain Nidāna and Saṃprāpti of Āmavāta
2. Describe the Sāmānya lakṣaṇās and Pravṛddha lakṣaṇās of Āmavāta
3. Explain the types of Āmavāta

Long Answer Questions

1. Explain Āmavāta in detail

TOPIC 46

Vātarakta

Introduction

Vātarakta is one of the Vāta Vyādhi as well as one of the Rakta Pradoṣaja vikāra. Ācārya Caraka explained this disease in a separate chapter because of its Viśiṣṭa Nidāna (specificity of the cause), Viśiṣṭa Saṃprāpti (specific pathological manifestation of the disease) and Viśeṣa Kriyā (specific management) manifests due to the combination of vitiated Vāta and Rakta having characters similar to air and fire. Vātaśoṇita, Khuḍaṃ, Vātabalāsaka and Āḍhyavāta are the synonyms of Vātarakta.

Nirūkti and Paribhāṣā

- वातरक्ताभ्यां जनितो व्याधिः वातरक्तं । (Ca.Ca.Ci.29/1)

The disease manifested by Vāta and Rakta is Vātarakta.

- वातदूपितं रक्तं यत्र । (SKD)

The aggravated Vāta vitiating the Rakta is Vātarakta.

- येन वातरक्तं वातस्य रक्तस्य च दुष्ट्या जायते । (Ga.Su.Ni.1/25-29)
- वातरक्तं हि दुष्टेन वातेन दुष्टेन रक्तेन च विशिष्टसम्प्राप्तिकं विकारान्तरमेव । (Ma.M.N.23/1-3)

The disease manifested by Duṣṭa Vāta and Duṣṭa Rakta is Vātarakta.

- वायुर्विवृद्धो वृद्धेन रक्तेनावारितः पथि॥ कृत्स्नं सन्दूपयेद्रक्तं तज्ज्ञेयं वातशोणितम्। (Ca.Ci.29/10)

The aggravated Vāta gets obstructed in its course by aggravated Rakta; the obstructed Vāta in turn vitiates the Rakta resulting in the condition called Vātaśoṇita.

- तत् सम्पृक्तं वायुना दूषितेन तत्प्राबल्यादुच्यते वातरक्तम् । (Su.Ni.1/44)

The aggravated Rakta combines with vitiated Vāta and due to its predominance it is known as Vātarakta.

Synonyms

- खुडं वातबलासाख्यमाढ्यवातं च नामभिः॥ (Ca.Ci.29/11)
- आढ्यरोगं खुडं वातबलासं वातशोणितम्। तदाहुर्नामभिः..... ॥ (A.H.Ni.16/4)

Khuḍaṃ, Vātabalāsā and Āḍhyavāta are the synonyms of the disease Vātarakta.

- **Khuḍaṃ :** The word Khuḍaṃ indicates Sandhi (joints). Here, the vitiated doṣas afflicts mainly the small joints. Hence it is named as Khuḍaṃ.
- **Vātabalāsā :** In Vātarakta, strength of the Vāta gets increased due to being obstructed by Rakta. Hence it is named as Vātabalāsā.
- **Āḍhyavāta :** Usually, this condition afflicts the Āḍhya (rich) persons, hence it is termed as Āḍhyavāta.

Nidāna and Saṃprāpti

- प्रायशः सुकुमाराणां मिथ्याऽऽहारविहारिणाम् । रोगाध्वप्रमदामद्यव्यायामैश्चातिपीडनात् ॥
  ऋतुसात्म्यविपर्यासात् स्नेहादीनां च विभ्रमात् । अव्यवाये तथा स्थूले वातरक्तं प्रकुप्यति ॥ (Su.Ni.1/40-41)

Vātarakta occurs usually in the person who is Sukumāra (delicate constitution), who indulges in Mithyāhāravihāra (improper diet and activities), who suffer from Atipīḍita roga (excessive sickness), Atipīḍita adhva (excessive walking), Atipīḍita pramadā (excessive indulgence in women), Atipīḍita madya (excessive intake of alcohol), Atipīḍita vyāyāma (excessive exercise), Ṛtusātmyaviparyāsāt (who lead a life opposite to that indicated for a particular seasons), Snehādīnāṃ ca vibhramāt (who gets faulty oleation etc. procedures), Avyavāya (who abstain from sexual intercourse) and Sthūla (who are obese).

- लवणाम्लकटुक्षारस्निग्धोष्णाजीर्णभोजनैः। क्लिन्नशुष्काम्बुजानूपमांसपिण्याकमूलकैः॥
  कुलत्थमाषनिष्पावशाकादिपललेक्षुभिः। दध्यारनालसौवीरशुक्ततक्रसुरासवैः॥
  विरुद्धाध्यशनक्रोधदिवास्वप्नप्रजागरैः। प्रायशः सुकुमाराणां मिष्टान्नसुखभोजिनाम् ॥
  अचङ्क्रमणशीलानां कुप्यते वातशोणितम्। अभिघातादशुद्ध्या च प्रदुष्टे शोणिते नृणाम्॥
  कषायकटुतिक्ताल्परूक्षाहारादभोजनात्। हयोष्ट्रयानयानाम्बुक्रीडाप्लवनलङ्घनैः ।
  उष्णे चात्यध्ववैषम्याद्व्यवायाद्वेगनिग्रहात् । वायुर्विवृद्धो वृद्धेन रक्तेनावारितः पथि॥
  कृत्स्नं सन्दूषयेद्रक्तं तज्ज्ञेयं वातशोणितम्। (Ca.Ci.29/5-10)

The Vāta and Rakta gets aggravated by indulging in excessive intake of Lavaṇa kaṭu kṣāra snigdha bhojana (saline, sour, pungent, alkalies and unctuous food substances), Uṣṇa bhojana (hot food), Ajīrṇa bhojana (uncooked foods), Klinnaśuṣka ambujānūpa māṃsa (intake of putrefied and dried meat of aquatic and marshy land animals), Piṇyāka (oil cake), Mūlaka (radish), Kulattha (red gram), Māṣa (black gram), Niṣpāva, Śākādi (leafy vegetables), Palala (sesamum paste), Ikṣu (sugarcane), Dadhi (curd), Aranāla (sour gruel), Sauvīraka (fermented drink), Śukta (vinegar), Takra (buttermilk), Surā (alcohol) and Āsava; intake of Viruddhāśana (incompatible diets), Adhyāśana (eating food before the digestion of previous food), Krodha (anger), Divāsvapna (day sleep), Prajāgaraṇa (vigil), Sukumārāṇām (delicate constitution), Miṣṭānnasukhabhojināṃ (intake of sweet and delicious food substances) and Acaṅkramaṇa śīlānāṃ (not used to walking).

Besides, the Rakta dhātu gets aggravated due to Abhighāta (injury) and Aśuddhyā (non cleanliness or improper purification of the body). While the Vāta aggravated by indulging in Kaṣāyakaṭutiktālparūkṣa āhāra (astringent, pungent, bitter and non-unctuous food substances), Abhojanāt (starvation), Hayoṣṭrāyaṇā (riding over horse and camel), Ambukriḍā (playing in the water), Plavana (swimming), Laṅghanaiḥ (fasting), Uṣṇa cātyādhva vaiṣamyāt (excessive walking during hot season), Vyavāya (excessive sexual intercourse) and Veganigrahān (suppression of natural urges).

Due to the above said etiological factors, the Vāta aggravates and gets obstructed in its passage by aggravated Rakta; affects the entire Rakta resulting in the condition called Vātaśoṇita.

Samprāpti Ghaṭaka

Doṣa	Vāta, Pitta
Dūṣya	Rakta
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Agnimāndhya janya Āma
Srotas	Rakta vaha
Srotoduṣṭi	Saṅga
Udbhava sthāna	Āmapākvāśaya
Sañcāra sthāna	Rakta vaha
Vyakta sthāna	Sarva Sandhi
Rogamārga	Madhyama

Pūrvarūpa of Vātaraka

- ◉ स्वेदोऽत्यर्थं न वा कार्ष्ण्यं स्पर्शाज्ञत्वं क्षतेऽतिरुक्। सन्धिशैथिल्यमालस्यं सदनं पिडकोद्गमः॥
जानुजङ्घोरुकट्यंसहस्तपादाङ्गसन्धिषु। निस्तोदः स्फुरणं भेदो गुरुत्वं सुप्तिरेव च॥
कण्डूः सन्धिषु रुग्भूत्वा भूत्वा नश्यति चासकृत्। वैवर्ण्यं मण्डलोत्पत्तिर्वातासृक्पूर्वलक्षणम्॥ (Ca.Ci.29/16-18)

Svedo atyartham na (excessive or absence of perspiration), Kārṣṇya (blackishness), Sparśajñatvaṃ (loss of tactile sensation), Kṣate atiruk (severe pain as if injured), Sandhi śaithilya (laxity of joints), Ālasya (lassitude), Sadana (malaise), Piḍakodgama (appearance of small eruptions), Janujaṅgorukaṭyaṃsahastapāda aṅgasandhiṣu nistodaḥ spuraṇa bhedo gurutvaṃ suptireva (pricking pain, twitching pain, piercing pain, heaviness and numbness of knee, shanks, thigh, waist, shoulders, hand, legs and other joints of the body), Kaṇḍūḥ sandhiṣu (itching sensation in the joints), Rukbhūtvā bhūtvā naśyati cāsakṛt (appearance and disappearance of pain in the joints), Vaivarṇyaṃ (discoloration) and Maṇḍalotpatti (appearance of round patches) are the premonitory symptoms of Vātarakta.

- ◉ प्राग्रूपे शिथिलौ स्विन्नौ शीतलौ सविपर्ययौ । वैवर्ण्यतोदसुप्तत्वगुरुत्वौषसमन्वितौ ॥ (Su.Ni.1/47)

The premonitory symptoms of Vātarakta are Śithilā (laxity of joints), Svinnā (perspiration), Śītala (coldness) as well as their Viparyayā (opposite to all of these) in association with Vaivarṇya (discoloration), Toda (pricking pain), Suptatva (numbness), Gurutva (heaviness) and Oṣa (burning sensation in the feet).

Sthāna of Vātarakta

- ◉ तस्य स्थानं करौ पादावङ्गुल्यः सर्वसन्धयः। कृत्वाऽऽदौ हस्तपादे तु मूलं देहे विधावति॥ (Ca.Ci.29/12)

Sthāna (location) of Vātarakta are Kara (hands), Pāda (feet), Aṅguli (fingers) and Sarvasandhi (all joints). At first it establishes its roots in hands and feet (Hastapāda mūlaṃ) and then spread all over the body.

- ◉ पादयोर्मूलमास्थाय कदाचिद्धस्तयोरपि । आखोर्विषमिव क्रुद्धं तद्देहमनुसर्पति ॥ (Su.Ni.1/48)

The disease spreads all over the body like a rat poison beginning from the Pāda mūla (sole of the feet) or sometimes from the hands.

Bheda and Lakṣaṇās of Vātarakta

Sthānika Bheda

- ◉ उत्तानमथ गम्भीरं द्विविधं तत् प्रचक्षते। त्वङ्मांसाश्रयमुत्तानं गम्भीरं त्वन्तराश्रयम्॥ (Ca.Ci.29/19)

Vātarakta is classified into two types namely Uttāna and Gambhīra. Uttāna (superficial) is located at Tvak (skin) and Māṃsa (muscles), while Gambhīra (deep) is located in Tvantarā (deeper dhātus).

Uttāna Vātarakta

- ◉ कण्डूदाहरुगायामतोदस्फुरणकुञ्चनैः। अन्विता श्यावरक्ता त्वग्बाह्ये ताम्रा तथेष्यते॥ (Ca.Ci.29/20)

Kaṇḍū (itching), Dāha (burning), Ruk (pain), Āyāma (stretching), Toda (pricking pain), Sphuraṇa (twitching), Akuñjana (constriction) associated with Śyāvarakta tvagbāhye tāmrā (skin becomes blackish, red or coppery colour) are the clinical features of Uttāna Vātarakta.

Gambhīra Vātarakta

◉ गम्भीरे श्वयथुः स्तब्धः कठिनोऽन्तर्भृशार्तिमान्। श्यावस्ताम्रोऽथवा दाहतोदस्फुरणपाकवान्॥
रुग्विदाहान्वितोऽभीक्ष्णं वायुः सन्ध्यस्थिमज्जसु। छिन्दन्निव चरत्यन्तर्वक्रीकुर्वंश्च वेगवान्॥
करोति खञ्जं पङ्गुं वा शरीरे सर्वतश्चरन्। सर्वैर्लिङ्गैश्च विज्ञेयं वातासृगुभयाश्रयम्॥ (Ca.Ci.29/21-23)

In Gambhīra Vātarakta, there is Śvayathu (oedema), Stabdha (stiffness), Kaṭhino (hardness), Antarbhṛśārtimān (severe pain inside), Śyāvastāmrāthavā (blackish or coppery colour), Dāha (burning), Toda (pricking pain), Sphuraṇa (twitching) and Pākavān (suppuration); when Vāta reaches Sandhi (joints), Asthi (bones) and Majjā (bone marrow) it causes Ruk (severe pain) and Vidāha (burning sensation); the forcefully moving Vāta produces cutting type of pain and deformities of the joints and it may even makes the person lame. If all the features of both Uttāna and Gambhīra Vātarakta are present and then one should considered it as Ubhayāśrita Vātarakta.

Doṣaja bheda

◉ तत्र वातेऽधिके वा स्याद्रक्ते पित्ते कफेऽपि वा। संसृष्टेषु समस्तेषु यच्च तच्छृणु लक्षणम्॥ (Ca.Ci.29/24)

Vātarakta is classified into eight types namely Vātaja, Raktaja, Pittaja, Kaphaja, Saṃsargaja and Sannipātaja.

Vātaja Vātarakta

◉ विशेषतः सिरायामशूलस्फुरणतोदनम् । शोथस्य कार्ष्ण्यं रौक्ष्यं च श्यावतावृद्धिहानयः॥
धमन्यङ्गुलिसन्धीनां सङ्कोचोऽङ्गग्रहोऽतिरुक्। कुञ्चनस्तम्भने शीतप्रद्वेषश्चानिलेऽधिके॥ (Ca.Ci.29/25-26)

◉ वातेऽधिकेऽधिकं तत्र शूलस्फुरणतोदनम्। शोफस्य रौक्ष्यकृष्णत्वश्यावतावृद्धिहानयः॥
धमन्यङ्गुलिसन्धीनां सङ्कोचोऽङ्गग्रहोऽतिरुक्। शीतद्वेषानुपशयौ स्तम्भवेपथुसुप्तयः॥ (A.H.Ni.16/12-13)

Sirāyāma (dilatation of the vessels), Śūla (pain), Sphuraṇa (twitching pain), Todanam (pricking pain), Śothasya kārṣṇyam (blackness of oedema),Raukṣyaṃ (non-unctuousness), Śyāvatā (blackish discoloration), Vṛddhi hānayaḥ (sudden increase or decrease of the disease), Dhamanyaṅgulisandhīnāṃ saṅkoca (contractures of vessels, fingers and joints), Aṅga graham (stiffness of body parts), Atiruk (severe pain), Kuñcana (contracture), Stambhana (stiffness), Vepathu (tremors), Suptayaḥ (numbness), and Śītapradveṣa (aversion to cold substances) are the clinical features of Vātaja Vātarakta.

Raktaja Vātarakta

◉ श्वयथुर्भृशरुक् तोदस्ताम्रश्चिमिचिमायते। स्निग्धरूक्षैः शमं नैति कण्डूक्लेदान्वितोऽसृजि ॥ (Ca.Ci.29/27)

◉ रक्ते शोफोऽतिरुकतोदस्ताम्रश्चिमिचिमायते। स्निग्धरूक्षैः शमं नैति कण्डूक्लेदसमन्वितः॥ (A.H.Ni.16/14)

Śvayathurbhṛśaruk (swelling with intense pain), Toda (pricking pain), Tāmrā (coppery discoloration), Cimicimāyate (tingling sensation), Snigdharūkṣaiḥ śamaṃ neti (not pacified either by unctuous or non-unctuous therapy) associated with Kaṇḍū (itching sensation) and Kleda (moistureness) are the clinical features of Raktaja Vātarakta.

Pittaja Vātarakta

◉ विदाहो वेदना मूर्च्छा स्वेदस्तृष्णा मदो भ्रमः। रागः पाकश्च भेदश्च शोषश्चोक्तानि पैत्तिके॥ (Ca.Ci.29/28)

◉ पित्ते विदाहः सम्मोहः स्वेदो मूर्च्छा मदः सतृट्। स्पर्शाक्षमत्वं रुग्रागः शोफः पाको भृशोष्मता॥ (A.H.Ni.16/15)

Vidāha (burning sensation after meals), Vedanā (pain), Mūrcchā (fainting), Sveda (perspiration), Tṛṣṇā (thirst), Mada (intoxication), Bhrama (giddiness), Sparśākṣamatvaṃ (inability to tolerate touch), Rāga (reddish discoloration), Pāka (suppuration), Bheda (bursting) and Śoṣa (dryness) are the clinical features of Pittaja Vātarakta.

Kaphaja Vātarakta

◉ स्तैमित्यं गौरवं स्नेहः सुप्तिर्मन्दा च रुक् कफे। (Ca.Ci.29/29)

◉ कफे स्तैमित्युगुरुतासुप्तिस्निग्धत्वशीतताः। कण्डूर्मन्दा च रुक् .....॥ (A.H.Ni.16/16)

Staimitya (as if covered with wet cloth), Gaurava (heaviness), Snehaḥ (unctuousness), Śītatā (coldness), Supti (numbness), Kaṇḍū (itching), and Mandā ruja (mild pain) are the clinical features of Kaphaja Vātarakta.

Saṃsarja and Sannipātaja Vātarakta

◉ हेतुलक्षणसंसर्गाद्विद्याद्द्वन्द्वत्रिदोषजम्॥ (Ca.Ci.29/29)

◉ ....द्वन्द्वसर्वलिङ्गं च सङ्करे। (A.H.Ni.16/16)

The clinical features of two doṣas or three doṣas are associated with combined etiological factors and symptoms.

Upadrava (complications)

◉ अस्वप्नारोचकश्वासमांसकोथशिरोग्रहाः। मूर्च्छायमदरुक्तृष्णाज्वरमोहप्रवेपकाः॥
हिक्कापाङ्गुल्यवीसर्पपाकतोदभ्रमक्लमाः। अङ्गुलीवक्रता स्फोटा दाहमर्मग्रहार्बुदाः॥
एतैरुपद्रवैर्वर्ज्यं मोहेनैकेन वाऽपि यत्। सम्प्रस्रावि विवर्णं च स्तब्धमर्बुदकृच्च यत्॥
वर्जयेच्चैव सङ्कोचकरमिन्द्रियतापनम्। अकृत्स्नोपद्रवं याप्यं साध्यं स्यान्निरुपद्रवम्॥ (Ca.Ci.29/31-34)

Asvapna (insomnia), Arocaka (anorexia), Śvāsa (dyspnoea), Māṃsakotha (putrefaction of muscle), Śirograhā (stiffness of the head), Mūrcchā (fainting), Mada (intoxication), Ruk (pain), Tṛṣṇā (thirst), Jvara (fever), Moha (delusion), Pravepakā (tremors), Hikkā (hiccup), Paṅgu (lameness), Visarpa (erysipelas), Pāka (suppuration), Toda (pricking pain), Bhrama (giddiness), Klamā (exhaustion), Aṅguli vakratā (deformity of fingers), Sphoṭā (blisters), Dāha (burning sensation), Marma graha (stiffness of vital parts) and Arbuda (tumours) are the complications of Vātarakta which makes the disease as Asādhya (incurable). However, only one Upadravā, i.e. Moha (delusion) is enough to make the disease incurable.

The disease associated with Saṃprasrāvi (profuse discharge from the lesion), Vaivarṇya (discoloration), Stabdha (stiffness), Arbuda (becomes tumour), Saṅkoca (contracture), and Indriyatāpanam (deranged sense organs) should be rejected.

If all the above complications are not present, then the disease is Yāpya (palliable) and that absence of complications is Sādhya (curable).

Sādhyāsādhyata (prognosis)

- एकदोषानुगं साध्यं नवं, याप्यं द्विदोषजम्। त्रिदोषजमसाध्यं स्याद्यस्य च स्युरुपद्रवाः॥ (Ca.Ci.29/30)

The Vātarakta caused by one doṣa and of recent onset is Sādhya (curable), that caused by two doṣas are Yāpya (palliable) and the one caused by all the three doṣas are Asādhya (incurable). That which is associated with Upadravā (complications) is also Asādhya (incurable).

In Contemporary science,

Vātarakta can be correlated with multiple disorders such as gouty arthritis, rheumatoid arthritis, psoriatic arthritis, peripheral vascular diseases etc.

In general, Vātarakta can be correlated with Gouty arthritis and Rheumatoid arthritis due to presence of similar clinical presentations. But there is difference of opinion regarding this among the Ayurvedic scholars. Some consider Āmavāta as rheumatoid arthritis and Vātarakta as gouty arthritis. However, gouty arthritis shows the clinical features of Pittaja Vātarakta and rheumatoid arthritis shows the clinical features of Kaphaja Vātarakta.

Based on the Avasthā Viśeṣa of Vātarakta (i.e. Uttāna and Gambhīra), it can be correlated with Psoriatic arthritis and based on the Āvaraṇa Saṃprāpti i.e. pathogenesis involving occlusion by Rakta or Kapha and Medas with aggravation of Vāta and Rakta, it can be correlated with peripheral vascular diseases such as Thrombo angitis obliterens (TAO or Buerger's disease), varicose veins, varicose ulcers, deep vein thrombosis etc.

Difference between Sandhigatavāta, Āmavāta and Vātarakta

Factors	Sandhigatavāta	Āmavāta	Vātarakta
Nidāna	Vāta Prakopaka Nidāna	Āma Prakopaka and Vāta Prakopaka Nidāna	Vāta and Rakta Prakopaka Nidāna
Doṣa Pradhāna	Vāta Pradhāna	Vāta Pradhāna Tridoṣa	Vāta, Pitta
Dūṣya	Asthi, Majjā and Snāyu	Rasa, Māṃsa, Asthi, Majjā, Sandhi, and Snāyu	Rakta
Pūrvarūpa	Avyakta Lakṣaṇa	Śirorujā and Gātrarujā	Śithilā, Svinnā, Śītala, Saviparyayā, Vaivarṇya, Toda, Suptatva, Gurutva and Oṣa
Śūla	Vātavat	Vṛścika vyāviddha	Ākho viṣa
Sthāna	Sarva Sandhi	Sarva Sandhi	Kara, Pāda, Aṅguli and Sarvasandhi
Anya lakṣaṇās	Prasāraṇākuñjanayo Pravṛtti Savedanā Vātapūrṇādṛtisparśa śotha, Hanti sandhīn, Sandhi śūla and Sandhi śopha	Aṅgamarda, Aruci, Tṛṣṇā, Ālasya, Gaurava, Jvara, Apāka and Aṅga śūnam	Śvayathu, Stabdha, Kaṭhino, Antarbhṛśārtimān, Śyāvastāmrāthavā, Dāha, Toda, Sphuraṇa and Pākavān
Upaśaya	Vāluka Svedana	Abhyaṅga	Rakta śodhana

Short Answer Questions

1. Explain Nidāna and Saṃprāpti of Vātarakta
2. Describe Uttāna and Gambhīra Vātarakta
3. Write the differences between Sandhigatavāta, Āmavāta and Vātarakta

Long Answer Questions

1. Define Vātarakta and explain the Nidāna, Saṃprāpti, Bheda and Lakṣaṇās of Vātarakta

TOPIC 47

Immunological and Metabolic Disorders

Introduction

Immuno-metabolic disorders represent a group of conditions characterized by the intersection of immune system malfunction and metabolic disturbances, resulting in chronic inflammation and tissue damage. Notable examples include metabolic syndrome, type 1 and 2 diabetes, rheumatoid arthritis, obesity-related immune dysfunction, and autoimmune thyroiditis. These disorders exhibit key characteristics, such as chronic inflammation, immune cell dysregulation, metabolic disruptions (insulin resistance, dyslipidemia), oxidative stress, mitochondrial dysfunction, epigenetic alterations, and changes in the gut microbiome. This section will focus specifically on the immuno-metabolic aspects of rheumatic conditions, including Rheumatic fever, Rheumatoid arthritis, Systemic Lupus Erythematosus (SLE), Ankylosing spondylitis, and Gout.

RHEUMATIC FEVER (RF)

Introduction

Rheumatic Fever (RF) is a systemic, acute inflammatory disease that occurs following a group A streptococcal infection (GAS), typically after strep throat (a type of pharyngitis) or scarlet fever. RF is characterized by various combinations of joint pain and swelling, cardiac valvular regurgitation with the potential for secondary heart failure, chorea, skin and subcutaneous manifestations and fever. Long-term consequences of RF include damage to heart valves, leading to Rheumatic Heart Disease (RHD), often resulting from recurrence of Rheumatic Fever. Due to this close relationship, Rheumatic Fever and Rheumatic Heart Disease are frequently discussed together in epidemiological contexts. Globally, the incidence of Rheumatic Heart Disease, a complication of Rheumatic Fever, increased by 49.7% since 1990, with approximately 2.79 million new cases reported in 2019. India bears the highest burden of Rheumatic Heart Disease, accounting for around 27% of all cases worldwide. The country reports a significant number of Rheumatic Fever cases, predominantly among children and young adults.

Pathophysiology

Rheumatic Fever is caused by Group A Streptococcus (GAS) infection, leading to an autoimmune response affecting various organs including the valvular endothelium of the heart valves, synovial tissue in the joints, basal ganglia in the brain, and subcutaneous tissue in the skin. Valve damage can result in valvular regurgitation or stenosis, ultimately leading to the onset of Rheumatic Heart Disease, which is characterized by persistent inflammation and scarring. Key contributors to this process include CD4+ T cells, macrophages, antibodies (IgG, IgM), cytokines (TNF-α, IL-6, IL-1β), and adhesion molecules (ICAM-1, VCAM-1). Pathological features include Aschoff bodies (granulomatous lesions), valve fibrosis and scarring, joint inflammation and synovitis, and skin manifestations (erythema marginatum). The immunological and molecular mechanisms involve molecular mimicry, epitope spreading, immune cross-reactivity, and cytokine dysregulation. Genetic predisposition, such as the HLA-DRB1*07:01 allele, also increases susceptibility to Rheumatic Fever.

Clinical features:

The clinical features of Rheumatic Fever typically develop 14 to 28 days after a Group A Streptococcal infection, such as strep throat or scarlet fever, often affecting multiple organ systems. The symptoms include fever, polyarthritis (migratory joint pain), carditis (inflammation of the heart), fatigue, sydenham chorea (involuntary, purposeless movements), erythema marginatum (a flat or slightly raised, painless skin rash) and subcutaneous nodules.

Diagnosis:

The diagnosis of Rheumatic Fever is clinically based on the revised Jones criteria, throat swab culture, blood investigations and cardiac evaluation. The revised Jones Criteria for Diagnosing Rheumatic Fever are

Major Criteria	Carditis (clinical or subclinical)
	Polyarthritis
	Chorea
	Subcutaneous nodules
	Erythema marginatum

Minor Criteria	Polyarthralgia
	Fever ≥38.5° C
	ESR (erythrocyte sedimentation rate) >60 mm/h and/or CRP (C-reactive protein) >3.0 mg/dL
	ECG with prolonged PR interval

The presence of 2 major criteria or 1 major and 2 minor criteria is required for the diagnosis of an initial episode of Rheumatic Fever.

Throat swab culture, specifically Nucleic acid amplification tests (NAAT), is considered the gold standard for detecting Group A Streptococcus infection due to its increased sensitivity and capacity to identify lower bacterial loads.

Blood investigations may indicate elevated Antistreptolysin O (ASO) or antideoxyribonuclease B (ADB) antibody titers, increased erythrocyte sedimentation rate, and elevated C-reactive protein levels. Furthermore, comprehensive cardiac evaluations, including chest radiography, electrocardiography (ECG), and echocardiography, are imperative for all patients with Rheumatic fever. Cardiac imaging may reveal cardiomegaly and pulmonary oedema, while ECG findings may exhibit a prolonged PR interval and echocardiographic assessments may disclose signs of valvulopathy or heart failure.

RHEUMATOID ARTHRITIS (RA)

Introduction

Rheumatoid arthritis (RA) is an autoimmune and chronic inflammatory disorder primarily affecting the peripheral joints in a symmetrical manner, producing inflammation of joints, destructing the cartilage and deforming the bones associated with systemic involvement. The global prevalence of Rheumatoid arthritis has increased by 7.4% from 1990 to 2017, affecting an estimated 17.6 million individuals worldwide. In India, the prevalence of Rheumatoid Arthritis (RA) is estimated to range from 0.7% to 1% of the population, with a higher incidence in women than men, typically manifesting in the fourth or fifth decade of life.

The cause of Rheumatoid arthritis is unknown. As an autoimmune disease, both genetic and environmental factors play an important role in the manifestation of the disease. Genetic predisposition is evident with a 2 to 4-fold increased risk among first-degree relatives. Notably, the presence of genes HLA-DR4 and HLA-DR1 has been linked to the disease in the Indian population. Furthermore, environmental determinants such as climatic influences, urbanization, gender disparities, tobacco usage, and specific microbial agents and their components are implicated in the disease's manifestation.

Pathophysiology:

The pathology of Rheumatoid arthritis is characterized by the infiltration of inflammatory cells, such as macrophages, lymphocytes and plasma cells, into the synovial tissue around the joint. As a result, it becomes inflamed and oedematous. As the disease progresses, synovial tissue hyperplasia will form with fibrosis and granular tissue over a joint surface, i.e., pannus tissue, which eventually replaces the articular cartilage. Synovial infiltrates show plenty of lymphocytes, mainly CD4+ T cells. The immune system produces pro-inflammatory cytokines (IL-1, IL-12, IL-18, tumour necrosis factor-alpha (TNF-α), gamma-interferon (IFN-gamma), and granulocyte-macrophage colony-stimulating factor) and anti-inflammatory cytokines (IL4, IL-10, IL-13, IFN-alpha and transforming growth factor-beta) in the synovium. The imbalances between these two cytokines are the causes of acute synovial proliferation and long-term destructive changes in the cartilage and bone, and the systemic manifestations of Rheumatoid arthritis.

Clinical Features:

The onset of Rheumatoid arthritis is insidious, often beginning with low grade fever, malaise, arthralgia, anorexia, weakness, loss of weight and anaemia as premonitory symptoms before progressing to joint inflammation and swelling.

The clinical features of Rheumatoid arthritis are pain and swelling of small joints of hands and legs but larger joints like the shoulders, elbows, hips, knees and ankles can also be involved in a symmetrical pattern with morning stiffness of more than one-hour duration. As the disease progresses, there will be destruction in the tendon sheath and bursae leading to tenosynovitis and bursitis and destruction of joint resulting in joint instability and deformities.

Deformities appears in the form of

- **Z deformity** : Radial deviation at the wrist with ulnar deviation of the fingers is usually associated with anterior subluxation of metacarpophalangeal joints.
- **Swan neck deformity** : Hyperextension of the proximal interphalangeal joint with fixed flexion at distal interphalangeal joints.
- **Boutonniere deformity** : Fixed flexion at the proximal interphalangeal joint and hyperextension at the distal interphalangeal joint.

Extra-articular manifestations: Rheumatoid arthritis is a systemic disease and involves most of the systems in the body. Rheumatoid nodules develop over the bony prominences subjected to mechanical pressure like olecranon, scapula, Achilles tendon, fingers and occipital region associated with constitutional symptoms such as fatigue, malaise, feverish feeling, loss of appetite and weight loss.

- **Pulmonary**: Pleurisy, pleural effusion, interstitial fibrosis, pneumonitis
- **Cardiac**: Pericarditis, infective endocarditis, rheumatic fever

- **Neurological**: Neuropathy
- **Skin**: Palmar erythema, vasculitis
- **Renal**: involvement is rare. Nephrotic syndrome may be seen due to secondary Amyloidosis
- **Eyes**: Episcleritis, Scleritis,

Diagnostic criteria of Rheumatoid arthritis

American Rheumatism Association revised the criteria for diagnosis of Rheumatoid arthritis (1987).

- Morning stiffness in and around joints lasting at least for more than one hour
- Arthritis of at least 3 or more of 14 possible joint areas i.e. right or left PIP, MCP, wrist, elbow, knee, ankle and MTP joints with soft tissue swelling (observed by a physician).
- Arthritis of hand joints i.e. at least one joint area swollen in a wrist, MCP or PIP joint
- Symmetric arthritis (simultaneous involvement of the same joint areas on both side of the body)
- Rheumatoid nodules on bony prominence or extensor surfaces or in juxta articular regions (observed by a physician)
- Positive Serum Rheumatoid Factor (RF) and Anti-Cyclic Citrullinated Peptide Antibody (Anti-CCP)
- Radiographic changes which include erosions or unequivocal bony decalcification localized in or most marked adjacent to the involved joints in posteroanterior hand and wrist joints.

Guidelines for the criteria : for diagnosing Rheumatoid arthritis at least four of these criteria must be satisfied and the first four criteria must have been present for at least 6 weeks.

Recently in 2010, European League Against Rheumatism (EULAR) / American College of Rheumatology (ACR) recommended the following score based classification criteria to diagnose the Rheumatoid Arthritis

- **Number and site of involved joints**
  - large joint 0
  - 2–10 large joints 1
  - 1–3 small joints (with or without involvement of large joints) 2
  - 4–10 small joints (with or without involvement of large joints) 3
  - >10 joints (at least one small joint) 5

- **Serology (at least 1 test result is needed for classification)**
  - Negative Rheumatic Factor (RA) *and* negative Anti–cyclic citrullinated peptide (ACPA) 0
  - Low-positive RF *or* low-positive ACPA 2
  - High-positive RF *or* high-positive ACPA 3
- **Acute-phase reactants (at least one test result is needed for classification)**
  - Normal CRP *and* normal ESR 0
  - Abnormal CRP *or* normal ESR 1
- **Duration of symptoms**
  - <6 weeks 0
  - ≥6 weeks 1

Guidelines for the criteria : Using the 2010 ACR/EULAR classification criteria for Rheumatoid arthritis, diagnosis as definite Rheumatoid arthritis is based upon the presence of synovitis in at least one joint, with the absence of an alternative diagnosis that better explains the synovitis. Achievement of a total score of ≥6/10 is needed for the diagnosis of a patient as having definite Rheumatoid arthritis.

SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

Introduction

Systemic lupus erythematosus (SLE) is a rare, chronic, multisystem autoimmune disease characterized by inflammation and damage to various organs, including the skin, joints, kidneys, brain, lungs, heart, and blood vessels. Approximately 3.41 million people worldwide are affected, with an estimated global incidence of 5.14 per 100,000 person-years and prevalence of 43.7 per 100,000 persons. In contrast, India reports a relatively low prevalence, ranging from 3.2 per 100,000 people. Systemic lupus erythematosus predominantly affects women of childbearing age, with a female to male ratio of 9 to 1.

The etiology of systemic lupus erythematosus (SLE) remains obscure, with a complex interplay of genetic, immunological, endocrine, and environmental factors implicated in its development. Although the exact inheritance pattern is not apparent, strong genetic involvement is suggested by familial segregation and high concordance rates in identical twins, reported to be as high as 50%. Furthermore, several environmental triggers have been identified. Additionally, certain drugs such as procainamide, hydralazine, isoniazid, and anti-tumor necrosis factor (TNF) drugs, which have been linked to a lupus-like phenomenon through their impact on DNA demethylation and alteration of self-antigens.

Pathophysiology

The pathophysiology of Systemic Lupus Erythematosus involves a complex interaction of genetic, immunological, and environmental elements. The disease is characterized by a breakdown in self-antigen tolerance, leading to the production of autoantibodies and activation of immune cells. The release of cytokines, including interferon-alpha (IFN-α), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6), along with complement activation (C3, C4), amplifies the immune response, resulting in the deposition of immune complexes, inflammation, and tissue damage. Hormonal influences, particularly estrogen, contribute to increased autoantibody production and immune cell activation. Endothelial dysfunction and vascular damage elevate the risk of cardiovascular complications. Furthermore, impaired apoptosis and deficient clearance of apoptotic cells exacerbate autoantibody production, perpetuating the sustained self-directed immune response characteristic of Systemic Lupus Erythematosus.

Clinical Features:

The clinical presentation of Systemic Lupus Erythematosus is characterized by a wide array of symptoms and a highly variable disease course, marked by periods of exacerbation and remission. Common manifestations include fatigue, fever, weight loss, and lymphadenopathy.

Musculoskeletal involvement is prevalent in systemic lupus erythematosus, with arthritis, arthralgia, and myalgia affecting a majority of patients. Dermatological manifestations encompass the classical butterfly-shaped malar rash, discoid lupus, photosensitivity, alopecia, and oral and nasal ulcers. Renal involvement, particularly in lupus nephritis, poses a significant concern, ranging from mild to severe.

Neurological and psychiatric manifestations encompass seizures, psychosis, depression, and cognitive impairment. Cardiovascular and pulmonary complications are noteworthy, including pericarditis, pleurisy, and pulmonary embolism. Gastrointestinal symptoms, such as abdominal pain and nausea, are common. Ocular manifestations include conjunctivitis, uveitis, and retinal vasculitis. Hematological abnormalities are frequent, including anemia, leukopenia, lymphopenia, and thrombocytopenia. Vasculitis and thrombotic events can occur, augmenting the risk of organ damage.

Diagnosis

Diagnosis of Systemic Lupus Erythematosus (SLE) involves a combination of clinical criteria and serological tests. The American College of Rheumatology (ACR) first developed the SLE classification criteria in 1971 and revised them in 1982 and 1997.

Revised American Rheumatism Association Criteria for Diagnosis of (SLE) (1997)

1. **Malar rash:** Fixed erythema, flat or raised

2. **Discoid rash:** Erythematous raised patches with keratotic scaling
3. **Photosensitivity:** Skin rash due to unusual reaction to sunlight
4. **Oral ulcers:** Oral or nasopharyngeal ulcers observed by the physician
5. **Arthritis:** Non-erosive, involving 2 or more peripheral joints
6. **Serositis:** Pleuritis/pleural effusion, or pericarditis/pericardial effusion
7. **Renal disorder:** Persistent proteinuria >0.5 g/day or 3+, or cellular casts
8. **Neurologic disorder:** Seizures or psychosis
9. **Hematologic disorder:** Haemolytic anaemia or leucopenia
   - ◈ (<4000/µL) on two or more occasions or lymphopenia
   - ◈ (<1500/µL) on two or more occasions or thrombocytopenia
   - ◈ (<100,000/µL) in the absence of offending drugs
10. **Positive anti-nuclear antibody** (ANA) (in the absence of drugs known to be associated with 'drug-induced SLE')
11. **Positive SLE-specific antibodies** such as Anti-ds DNA or anti-Smith antibody and/or antiphospholipid antibody

Diagnosis of Systemic Lupus Erythematosus requires at least 4 of the 11 criteria, either serially or simultaneously.

Serological tests for lupus include positive antinuclear antibodies (ANA), positive antibodies to deoxyribonucleic acid (DNA) (anti-dsDNA), positive anti-Smith antibodies and positive antiphospholipid antibody. Additional laboratory assessments involve evaluating serum complement levels (C3, C4), typically diminished during active disease, notably in patients with active nephritis. Erythrocyte sedimentation rate (ESR) commonly exhibits elevation during active disease, while C-reactive protein levels may not necessarily be elevated; however, elevated levels may indicate infection and serositis.

Optional tests may encompass a complete blood count to identify hematologic irregularities, blood chemistry tests to assess renal and hepatic function, urinalysis to detect proteinuria and hematuria, and imaging studies such as chest X-ray and echocardiogram to evaluate cardiovascular and pulmonary involvement.

ANKYLOSING SPONDYLITIS

Introduction

Ankylosing Spondylitis (AS) is a chronic inflammatory disease predominantly impacting the spine, sacroiliac (SI) joints, peripheral joints, digits, and entheses (connective tissues). Key features of the disease include persistent back pain, progressive spinal stiffness, and potential fusion of vertebrae. Additionally, Ankylosing Spondylitis may give rise to extraarticular manifestations, including inflammatory bowel disease, acute anterior uveitis, and psoriasis.

Although the etiology of ankylosing spondylitis remains largely undetermined, a significant association exists between the disease's prevalence and the presence of human leukocyte antigen (HLA)-B27 within specific populations. Notably, among HLA-B27-positive individuals, the prevalence of ankylosing spondylitis is approximately 5-6%, while first-degree adult relatives who are HLA-B27 positive have a higher prevalence, ranging from 10-30%. In India, an estimated 1.65 million individuals have been diagnosed with Ankylosing Spondylitis, with an anticipated annual growth rate of 2.95%, projected to reach 2 million by 2028. Typically, onset occurs between ages 20 and 30, with a higher prevalence observed in men than women.

Pathophysiology:

The pathophysiology of Ankylosing Spondylitis involves a complex interplay between genetic, environmental, and immune factors. A strong genetic association exists with the presence of human leukocyte antigen (HLA)-B27, suggesting a predisposition to the disease. However, HLA-B27 alone does not trigger Ankylosing Spondylitis; other genetic and environmental factors contribute to its development.

In Ankylosing Spondylitis, the immune system mistakenly targets self-antigens, leading to chronic inflammation. Activated T-cells and macrophages produce pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-17 (IL-17), and interleukin-23 (IL-23). These cytokines promote inflammation, bone resorption, and new bone formation. This chronic inflammation can lead to various clinical manifestations such as enthesitis, spondylitis, and sacroiliitis. Additionally, bone formation and fusion result from the formation of bony growths between vertebrae, leading to spinal stiffness and fusion. The inflammatory process also affects the gut, with research suggesting a link between gut microbiome dysbiosis and Ankylosing Spondylitis.

Clinical Features:

The cardinal presentation of Ankylosing Spondylitis typically involves low back pain that radiates to the buttocks or thighs, typically accompanied by morning stiffness. Symptoms worsen with inactivity and improve with movement.

As the disease progresses gradually, it affects the entire spine, leading to restricted lumbar spine mobility, sacroiliac joint pain, inflexible spine and chest due to ankylosis, secondary vertebral osteoporosis leading to an increased risk of fractures, thoracic and cervical spine kyphosis, and fixed flexion contractures in the hips or knees. Additionally, peripheral manifestations may include Plantar fasciitis, Achilles tendinitis, tenderness at bony prominences (iliac crest, greater trochanter), enthesitis (inflammation at tendon insertions), and asymmetrical peripheral arthritis of hips, knees, ankles, and shoulders.

Notably, peripheral joint involvement precedes spinal symptoms in approximately 10% of cases, while 10% of patients experience childhood onset. Fatigue is prevalent due to chronic sleep disturbances, pain, systemic inflammation, and inflammatory cytokines' impact on the brain. Anterior uveitis is the most common extra-articular feature, occasionally preceding joint disease.

Diagnosis

Diagnosis of Ankylosing Spondylitis is based on clinical, imaging, and serological criteria. The Assessment of Spondyloarthritis International Society (ASAS) 2009 axial spondyloarthritis criteria are commonly used to diagnose Ankylosing Spondylitis. To fulfill the imaging criteria, patients must have radiographic or magnetic resonance imaging (MRI) evidence of sacroiliitis, along with at least one spondyloarthritis feature.

To meet the clinical criteria, patients must test positive for human leukocyte antigen B27 (HLA-B27) and have at least two spondyloarthritis features, including dactylitis, enthesitis of the heel, family history of spondyloarthritis, history of inflammatory back pain, arthritis, psoriasis, inflammatory bowel disease, uveitis, elevated C-reactive protein, or a good response to nonsteroidal anti-inflammatory drugs (NSAID's).

Serological tests typically reveal positive HLA-B27, elevated erythrocyte sedimentation rate (ESR), and elevated C-reactive protein (CRP).

GOUT

Introduction

Gout is an acute inflammatory arthritis characterized by the deposition of monosodium urate (MSU) crystals in tissues, primarily in and around joints, leading to recurrent acute or chronic arthritis due to hyperuricemia. The global prevalence of gout ranges from 1% to 4%, with advanced age and male gender being the primary risk factors.

The etiology of gout typically encompasses multifactorial elements, including genetic predisposition, medical comorbidities such as kidney disease and diabetes, dietary factors including purine-rich foods and alcohol, and specific medications like diuretics and immunosuppressants. In rare instances, a singular genetic defect may be responsible for causing gout, usually associated with other medical complications. Irrespective of the underlying trigger, elevated serum uric acid levels increase the risk of clinical gout in susceptible individuals.

Pathophysiology

The pathophysiology involves multiple factors and the process begins with hyperuricemia, elevated levels of uric acid in the blood, resulting from increased production or decreased excretion. When uric acid levels exceed the saturation point, MSU crystals form and accumulate in joints, triggering an inflammatory response. Neutrophils and macrophages are activated, releasing pro-inflammatory cytokines like interleukin-1β (IL-1β) and tumor necrosis factor-alpha (TNF-α). Additionally, the NLRP3 inflammasome, a multi-protein complex in immune cells, senses urate crystals and activates pro-inflammatory cytokines, particularly IL-1β. This amplifies the inflammatory response and leads to the characteristic symptoms of gout, such as severe pain and swelling.

Clinical features:

Gout is a chronic disease characterized by four distinct stages:

- **Asymptomatic Hyperuricemia:** Elevated serum urate levels increase the risk of acute gout attacks, although patients may remain asymptomatic.
- **Acute Gout Attack:** Sudden, severe pain and swelling occur in a single joint (typically the first metatarsophalangeal joint), with maximum inflammation within 12-24 hours and resolution within 3-14 days, even without treatment.
- **Intercritical Gout:** Following the acute attack, patients enter an asymptomatic stage, where hyperuricemia persists, and crystal deposition continues.
- **Chronic Tophaceous Gout:** Untreated or undertreated gout leads to gradual joint destruction and urate crystal deposits (tophi), causing chronic arthritis.

Approximately 60% of patients experience a second gout attack within one year, and 80% within three years. Gout attacks may be triggered by local trauma, alcohol consumption, dietary changes, and certain medications. Physical examination findings include erythematous, swollen, warm, and tender joints, as well as subcutaneous tophi deposits.

Diagnosis

The diagnosis of gout is based on clinical assessment, laboratory investigations, and imaging studies. Clinically, gout is characterized by sudden, severe joint pain, swelling, redness, and warmth, typically affecting the first metatarsophalangeal joint.

Laboratory tests encompass the evaluation of serum uric acid levels, synovial fluid analysis for monosodium urate crystals, complete blood count (CBC), blood chemistry, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). Elevated serum uric acid levels and increased ESR/CRP levels during acute attacks serve as key indicators of gout..

Imaging studies, such as X-rays, ultrasound, dual-energy computed tomography (DECT) scan, and magnetic resonance imaging (MRI), play a crucial role in identifying joint damage, tophi (deposits of urate crystals), and inflammation.

DIFFERENTIAL DIAGNOSIS OF ARTHRITIS

Arthritis, inflammation of the joints, requires a comprehensive differential diagnosis. Accurate diagnosis relies on age, sex, patient history, physical examination, joint distribution (monoarticular, oligoarticular, or polyarticular), symptom duration (acute or chronic), morning stiffness, systemic symptoms (fever, fatigue), extra-articular manifestation, co-morbid conditions, laboratory tests (rheumatoid factor, anti-CCP) and imaging studies (X-rays, ultrasound) The primary considerations include

- **Degenerative arthritis** includes Osteoarthritis (OA) and Degenerative Joint Disease (DJD), characterized by wear and tear on joints
- **Inflammatory arthritis** includes Rheumatoid Arthritis (RA), Psoriatic Arthritis (PsA), Ankylosing Spondylitis (AS), Reactive Arthritis (ReA), Lupus Arthritis, Spondyloarthritis (SpA), Gout, and Pseudogout. These conditions share similar symptoms, such as joint pain, swelling, and stiffness.
- **Infectious arthritis,** like Septic Arthritis, Lyme Arthritis, Tuberculous Arthritis, and Fungal Arthritis, requires prompt diagnosis and treatment.
- **Crystal-induced arthritis,** including Gout and Pseudogout, results from crystal deposits in joints.
- **Autoimmune arthritis,** such as RA, Lupus Arthritis, Sjögren's Syndrome, and Mixed Connective Tissue Disease, involves immune system dysfunction.
- **Metabolic arthritis,** encompassing Gout, Pseudogout, Hemochromatosis, and Wilson's Disease, stemming from metabolic disorders.
- **Traumatic arthritis,** including Post-traumatic OA and avascular necrosis, following joint injuries.
- **Other conditions,** such as Fibromyalgia, Polymyalgia Rheumatica, Tendinitis, and Bursitis, require consideration.

MCQ's

1. Which of the following is a common cause of rheumatic fever (RF)

a) Viral infection
b) Group A streptococcal infection
c) Bacterial infection of the liver
d) Fungal infection

2. What is a long-term consequence of rheumatic fever
   - a) Chronic kidney disease b) Rheumatic heart disease
   - c) Chronic liver disease d) Pulmonary fibrosis

3. What are Aschoff bodies
   - a) Granulomatous lesions found in the heart
   - b) Inflammatory cells in the liver
   - c) Fibrotic nodules in the lungs
   - d) Calcified plaques in the arteries

4. Which of the following clinical manifestations is NOT a major criterion for diagnosing Acute Rheumatic Fever
   - a) Carditis b) Chorea
   - c) Elevated C-reactive protein d) Subcutaneous nodules

5. Which of the following genes is linked to an increased risk of rheumatoid arthritis in the Indian population
   - a) HLA-B27 b) HLA-DR4
   - c) HLA-A2 d) HLA-DQ2

6. What is the primary characteristic of the pathology of rheumatoid arthritis
   - a) Infiltration of inflammatory cells into the synovial tissue
   - b) Deposition of amyloid plaques in the brain
   - c) Calcification of the arterial walls
   - d) Formation of granulomas in the lungs

7. Which deformity is characterized by hyperextension of the proximal interphalangeal joint with fixed flexion at the distal interphalangeal joints
   - a) Z deformity b) Swan neck deformity
   - c) Boutonniere deformity d) Heberden's nodes

8. What is a common extra-articular manifestation of rheumatoid arthritis
   - a) Nephritis b) Pleurisy
   - c) Hepatitis d) Retinopathy

9. Which of the following is a common neurological manifestation of systemic lupus erythematosus (SLE)
   - a) Epilepsy b) Psychosis
   - c) Multiple sclerosis d) Parkinson's disease

10. Which type of rash is characteristic of systemic lupus erythematosus (SLE)
   - a) Bullous rash b) Malar rash
   - c) Vesicular rash d) Pustular rash

11. Which laboratory test is commonly used to support the diagnosis of systemic lupus erythematosus (SLE)
   - a) Anti-nuclear antibody (ANA) test b) Complete blood count (CBC)
   - c) Liver function test (LFT) d) Serum creatinine

12. Which of the following is a common renal manifestation of systemic lupus erythematosus
   - a) Lupus nephritis b) Polycystic kidney disease
   - c) Renal artery stenosis d) Nephrolithiasis

13. Which genetic marker is strongly associated with ankylosing spondylitis (AS)
   - a) HLA-B27 b) HLA-DR4
   - c) HLA-DQ2 d) HLA-A1

14. What is a common extra-articular manifestation of ankylosing spondylitis (AS)
   - a) Diabetes mellitus b) Acute anterior uveitis
   - c) Hyperthyroidism d) Chronic kidney disease

15. Which clinical feature is characteristic of ankylosing spondylitis (AS)
   - a) Symmetrical joint involvement
   - b) Morning stiffness that improves with activity
   - c) Rapid onset of symptoms
   - d) Predominantly affects the cervical spine

16. Which of the following is NOT a typical clinical feature of ankylosing spondylitis (AS)
   - a) Restricted lumbar spine mobility b) Sacroiliac joint pain
   - c) Symmetrical peripheral arthritis d) Morning stiffness

17. What is the primary cause of the inflammatory response in gout
    - a) Deposition of calcium pyrophosphate crystals
    - b) Deposition of monosodium urate (MSU) crystals
    - c) Deposition of cholesterol crystals
    - d) Deposition of oxalate crystals
18. Which joint is most commonly affected during an acute gout attack
    - a) Knee joint
    - b) Elbow joint
    - c) First metatarsophalangeal joint
    - d) Shoulder joint
19. What is the term for the asymptomatic stage of gout characterized by elevated serum urate levels
    - a) Acute gout attack
    - b) Intercritical gout
    - c) Chronic tophaceous gout
    - d) Asymptomatic hyperuricemia
20. Which imaging technique is useful for identifying urate crystal deposits (tophi) in gout
    - a) Ultrasound
    - b) X-ray
    - c) Dual-energy computed tomography (DECT) scan
    - d) Magnetic resonance imaging (MRI)

Answer Keys

1	b	2	b	3	a	4	c	5	b	6	a	7	b	8	b	9	b	10	b
11	a	12	a	13	a	14	b	15	b	16	c	17	b	18	c	19	d	20	c

Short Answer Questions

1. Explain Rheumatic fever
2. Explain Rheumatoid arthritis
3. Explain Systemic Lupus Erythematosus
4. Explain Ankylosing spondylitis
5. Explain Gout

TOPIC 48

Klaibya and Vandhyatva

KLAIBYA

Introduction

The word Klaibya refers to impotence i.e. a man who is unable to perform sexual intercourse because of inability to achieve erection. Dhvajabhaṅga is one among the types of Klaibya which means bending or downfall of penis in general. Ācārya Caraka described Klaibya along with Yoni vyāpat cikitsā. Ācārya Suśruta described Klaibya in Kṣīṇabalīya Vājīkaraṇa cikitsā.

Nirūkti and Paribhāṣa

- क्लैब्यमिति ध्वजानुच्छ्रायः। (Ca.Sū.28/18-19)

  That which causes Dhvajānucchrāya (lack of penile erection) is called as Klaibya.

- क्लैब्यं स्त्रीष्वनुत्साहः । (Ma.M.N.5/22)

  That which causes Strīṣvanutsāha (absence of desire or excitement in the female) is known as Klaibya.

- क्लैब्यमिति सत्यपि ध्वजोत्थाने मैथुनाशक्तिः । (Ca.Sū.28/18-19)

  That which causes Maithunāśakti (inability to perform sexual act) even though there is Dhvajotthāna (penile erection) is called Klaibya.

- क्लीबः स्यात्सुरताशक्तस्तद् भावः क्लैब्यमुच्यते ॥ (B.P.U.1/2)
- क्लीबः स्यात् सुरताशक्तस्तद्भावः क्लैब्यमुच्यते ॥ (B.R.92/1)

That which causes Klībaḥ syātsuratāśakta (inability to perform sexual act) is called Klaibya.

- क्लैब्यं रत्यनभिमुखता। (In.A.S.Sū.19/9)

That which causes Ratyanabhimukhatā (aversion towards sexual act) is called Klaibya.

- क्लैब्यमिति षण्ढतेत्यर्थः। (Ḍa.Su.Sū.24/9)
- क्लीबः नपुंसकः। (Ca.Ca.Ci.23/130-135)

The disease Klaibya means Ṣaṇḍhata or Napuṃsaka (impotent or hermaphrodite)

- षण्ढता - स्त्रीगमनाशक्तित्वम्। (Aru.A.H.Sū.4/19-20)

That which causes Strigamana aśaktitva (inability to perform sexual act with female) is called Ṣaṇḍha.

- षण्ढता-अपत्यजननासामर्थ्यम्। (He.A.H.Sū.4/19-20)

That which causes Apathyajanana asāmarthya (incapability to produce progeny) is called Ṣaṇḍha.

- ध्वजभङ्गो मेढ्रस्यास्तब्धता ॥ (In.A.S.Sū.13/24)

That which causes Meḍhrasyāstabdhatā (lack of rigidity of penis) is called Dhvajabhaṅga.

Sāmānya Lakṣaṇās of Klaibya

- क्लैब्यं सम्पद्यते तस्य शृणु सामान्यलक्षणम्। सङ्कल्पप्रवणो नित्यं प्रियां वश्यामपि स्त्रियम्॥ न याति लिङ्गशैथिल्यात् कदाचिद्याति वा यदि। श्वासार्तः स्विन्नगात्रश्च मोघसङ्कल्पचेष्टितः॥ म्लानशिश्नश्च निर्वीजः स्यादेतत् क्लैब्यलक्षणम्। सामान्यलक्षणं ह्येतद्विस्तरेण प्रवक्ष्यते॥ (Ca.Ci.30/155-157)

Saṅkalpapravaṇo nitya priyāṃ striyam na yāti liṅgaśaithilyāt (persistent inability to perform sexual intercourses with his beloved, willing and submissive partner though having persistent desire, due to the lack of erection), Śvāsārtaḥ svinnagātraśca moghasaṅkalpaceṣṭitaḥ (if he tries, he gets more exhausted with dyspnoea and excess perspiration), Mlānaśiśnaśca (flaccidity of penis) and Nirbījaḥ (absence of sperms or lack of ejaculation) are the clinical features of Klaibya..

Sāmānya Nidāna and Saṃprāpti of Klaibya

- रेतोदोषोद्भवं क्लैब्यं ... । (Ca.Ci.30/153)

Klaibya is mainly caused by Retodoṣa (morbidities of semen). Hence the causative factors described for Śukra duṣṭi can be adopted here in Klaibya.

- अतिव्यवायाद्व्यायामादसात्म्यानां च सेवनात्। अकाले वाऽप्ययोनौ वा मैथुनं न च गच्छतः॥ रूक्षतिक्तकषायातिलवणाम्लोष्णसेवनात्। नारीणामरसज्ञानां गमनाज्जरया तथा॥

चिन्ताशोकादविस्रम्भाच्छस्त्रक्षाराग्निविभ्रमात्। भयात्क्रोधादभीचाराद्व्याधिभिः कर्शितस्य च॥ वेगाघातात् क्षताच्चापि धातूनां सम्प्रदूषणात्। दोषाः पृथक् समस्ता वा प्राप्य रेतोवहाः सिराः॥ शुक्रं सन्दूषयन्त्याशु.... ॥ (Ca.Ci.30/135-139)

- Ativyavāyād (excessive sexual intercourse)
- Vyāyāmād (excessive physical exercise)
- Asātmyānāṃ sevanāt (intake of unwholesome substances)
- Akāle maithuna (untimely coitus)
- Apyayoni maithuna (coitus in other than vagina)
- Maithunaṃ na ca gacchata (abstinence from sexual act)
- Rūkṣatiktakaṣāya sevanāt (intake of non-unctuous, bitter and astringent substances)
- Atilavaṇāmloṣṇa sevanāt (excessive saline, sour and hot substances)
- Nārīṇāmarasajñānāṃ (not having interest in women)
- Gamanājjarayā (coitus with old women)
- Cintā (worries)
- Śoka (grief)
- Avisrambhāt (lack of confidence)
- Śastrakṣārāgnivibhramāt (faulty application of instrument, alkali and cauterization)
- Bhayāt (fear)
- Krodha (anger)
- Abhīcārād (exorcism)
- Vyādhibhiḥ karśitasya (emaciated due to other diseases)
- Vegaghātāt (suppression of natural urges)
- Kṣatāt (injury)
- Dhātu saṃpradūṣaṇāt (morbid affliction of dhātus)

The aforementioned causative factors contribute to the individual or combined aggravation of doṣas. Upon reaching the Retovaha srotas, these factors vitiate the Śukra dhātu, thereby leading to the manifestation of the condition known as Klaibya.

Samprāpti Ghaṭaka of Klaibya

Doṣa	Vāta Pradhāna Tridoṣas and Mānasika doṣas (Rajas and Tamas)
Dūṣya	Rasādi Sapta dhātus
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhya janya Āma
Srotas	Rasa, Śukra and Manovaha
Srotoduṣṭi	Saṅga, Atipravṛtti and Vimārga gamana
Sañcāra sthāna	Sarva Rasāyanīs
Adhiṣṭhāna	Vṛṣaṇa and Meḍhra
Udbhava sthāna	Āmapakvāśaya and Hṛdaya
Vyakta sthāna	Meḍhra
Rogamārga	Bāhya and Madhyama

Klaibya Bheda

◉ बीजध्वजोपघाताभ्यां जरया शुक्रसङ्क्षयात्॥ क्लैब्यं सम्पद्यते.... ॥ (Ca.Ci.30/154-155)

Ācārya Caraka classified Klaibya into four types based on their etiopathogenesis namely Bījopaghāta, Dhvajabhaṅga, Jarayā and Śukrakṣaya.

Bījopaghātaja Klaibya Nidāna and Lakṣaṇa

◉ शीतरूक्षाल्पसङ्क्लिष्टविरुद्धाजीर्णभोजनात् । शोकचिन्ताभयत्रासात् स्त्रीणां चात्यर्थसेवनात्॥
अभिचारादविस्रम्भाद्रसादीनां च सङ्क्षयात्। वातादीनां च वैषम्यात्तथैवानशनाच्छ्रमात्॥
नारीणामरसज्ञत्वात् पञ्चकर्मापचारतः। बीजोपघाताद्भवति पाण्डुवर्णः सुदुर्बलः॥
अल्पप्राणोऽल्पहर्षश्च प्रमदासु भवेन्नरः। हृत्पाण्डुरोगतमककामलाश्रमपीडितः॥
छर्द्यतीसारशूलार्तः कासज्वरनिपीडितः। बीजोपघातजं क्लैब्यं ... ॥ (Ca.Ci.30/158-163)

Due to intake of Śīta (cold), Rūkṣa (non-unctuous), Saṅkliṣṭa (tarnished), Viruddha (contrary food substances), Ajīrṇa bhojanāt (uncooked food substances), Śoka (grief), Cintā (worries), Bhaya (fear), Trāsā (terror) and Strīnāṃ cātyarthasevanāt (excessive sexual intercourse with women), Abhicārā (exorcism), Avisrambhāt (suspicion on partner), Rasādīnām saṅkṣayāt (deficiency of Rasādi dhātus), Vatādīnaṃ vaiṣamyāt (vitiation of Vātādi doṣas), Anaśanāt (fasting), Śramāt (exhaustion), Nārīṇāmarasajñānāṃ (not having interest in women) and Pañcakarma apacārata (improper application of five purificatory therapies), Bījopaghāta (destruction of Bīja) occurs. And the person becomes Pāṇḍuvarṇa (pale), Durbala (debilitated), Alpa prāṇa (low vitality), Alpaharṣaśca pramadāsu (inadequate erection for young women), Hṛdroga (cardiac disorders), Pāṇḍuroga (anaemia), Tamaka (dyspnoea), Kāmalā (jaundice), Śramapīḍita (exhaustion), Chardi (vomiting), Atisāra (diarrhoea), Śūla (colic), Kāsa (cough) and Jvara (fever). This Klaibya is due to Bījopaghāta (destruction of Bīja).

Dhvajabhaṅgaja Klaibya Nidāna and Lakṣaṇa

◉ ...... ध्वजभङ्गकृतं शृणु॥
अत्यम्ललवणक्षारविरुद्धासात्म्यभोजनात्। अत्यम्बुपानाद्विषमात् पिष्टान्नगुरुभोजनात्॥
दधिक्षीरानूपमांससेवनाद्व्याधिकर्षणात्। कन्यानां चैव गमनादयोनिगमनादपि॥
दीर्घरोगां चिरोत्सृष्टां तथैव च रजस्वलाम्। दुर्गन्धां दुष्टयोनिं च तथैव च परिस्रुताम् ।
ईदृशीं प्रमदां मोहाद्यो गच्छेत् कामहर्षितः। चतुष्पदाभिगमनाच्छेफसश्चाभिघाततः॥
अधावनाद्वा मेढ्रस्य शस्त्रदन्तनखक्षतात्। काष्ठप्रहारनिष्पेषाच्छूकानां चातिसेवनात्॥
रेतसश्च प्रतीघाताद्ध्वजभङ्गः प्रवर्तते। (Ca.Ci.30/163-167)

Intake of Atyamlalavaṇakṣāraviruddhāsātmyabhojanāt (excessive sour, saline, alkaline, contrary and unwholesome food substances), Atyambu pāna (drinking excess water), Viṣamāśana (irregular meals), Piṣṭānna (cake and pastries), Guru bhojanāt (heavy food substances), intake of Dadhi (curd), Kṣīra (milk) and Ānūpamāṃsa (meat of marshy animals), Vyādhibhiḥ karṣaṇāt (emaciated due to other diseases), Kanyānāṃ gamanāt (coitus with unmarried girl), Ayoni gamanāt (coitus in other than vagina); due to Pramadā (negligence), Moha (ignorance or foolishness) and Kāma (lust or desire), having intercourse with woman who has Dīrgharoga (chronic diseases), Cirotsṛṣṭāṃ (left intercourse for long period), Rajasvalām (menstruating), Durgandha (foul smell emitting from the vagina), Duṣṭayoni (other vaginal disorders) and Parisrutām (excessive discharge); Catuspadā gamanāt (coitus with animals), Śephaścābhighāta (injury to penis), Adhāvanāt meḍhra (non cleanliness of the penis), Śastradantanakha kṣatāt (injury to the penis due to sharp instruments, teeth and nails); Kāṣṭhaprahāra (strike by staff), Niṣpeṣā (rubbing or pressing), Śūkānāṃ atisevanāt (excess use of penile applications for increasing its size) and Retaśca pratighātād (suppressing the semen) leads to the Dhvajabhaṅgaja Klaibya.

◉ श्वयथुर्वेदना मेढ्रे रागश्चैवोपलक्ष्यते॥ स्फोटाश्च तीव्रा जायन्ते लिङ्गपाको भवत्यपि।
मांसवृद्धिर्भवेच्चास्य व्रणाः क्षिप्रं भवन्त्यपि॥ पुलाकोदकसङ्काशः स्रावः श्यावारुणप्रभः।
वलयीकुरुते चापि कठिनश्च परिग्रहः॥ ज्वरस्तृष्णा भ्रमो मूर्च्छा च्छर्दिश्चास्योपजायते।
रक्तं कृष्णं स्रवेच्चापि नीलमाविललोहितम्॥ अग्निनेव च दग्धस्य तीव्रो दाहः सवेदनः।
बस्तौ वृषणयोर्वाऽपि सीवन्यां वङ्क्षणेषु च॥ कदाचित्पिच्छिलो वाऽपि पाण्डुः स्रावश्च जायते।
श्वयथुर्जायते मन्दः स्तिमितोऽल्पपरिस्रवः॥ चिराच्च पाकं व्रजति शीघ्रं वाऽथ प्रमुच्यते।
जायन्ते क्रिमयश्चापि क्लिद्यते पूतिगन्धि च॥ विशीर्यते मणिश्चास्य मेढ्रं मुष्कावथापि च।
ध्वजभङ्गकृतं क्लैब्यमित्येतत् समुदाहृतम्॥ एतं पञ्चविधं केचिद्ध्वजभङ्गं प्रचक्षते। (Ca.Ci.30/169-176)

Meḍhra śvayathu (swelling in the penis), Meḍhra vedanā (pain in the penis), Meḍhra rāga (redness in the penis), Tīvrā sphoṭā (severe blisters), Liṅgapākā (suppuration of penis), Māṃsavṛddhi Kṣipraṃ vraṇa bhavanti (ulcers develop quickly), Pulākodakasaṅkāśa (appearance of watery discharge like rice), Śyāvāruṇaprabha srāva (blackish or reddish discharge), Valayīkurute (penis is curved), Kaṭhinaśca parigraha (hardening of penile circumference). Besides these, they suffers from Jvara (fever), Tṛṣṇā (thirst), Bhrama (giddiness), Chardi (vomiting), Raktakṛṣṇanīlamavilalohita srāva (reddish, blackish, bluish and blood mixed discharges occur), Agnineva dagdhasya Tīvrādāhaḥ (burning sensation of penis as if burnt with fire) Savedanaḥ bastau vṛṣaṇayorvāapi sīvanyāṃ vaṃkṣaṇaṣu (with pain in the bladder, scrotum, perineal suture and groin regions); at times Picchilā vāapi pāṇḍuḥ srāva (slimy or pale discharge), Manda śvayathu stimitoalpaparisravaḥ (mild swelling and feeling as if covered with wet cloth and scanty discharge); Cirāt pāka vā śīghra pramucyate (and it takes long time to suppurate or gets subsides quickly), Krimayaścāpi jāyante (formation of maggots) with Klidyate (moist) and Pūtigandha (foul smell); Meḍhramaṇi viśīryate muṣkāvathāpi (necrosis of glans penis occurs or the whole penis along with scrotum may fall). This Klaibya is due to Dhvajabhaṅga (looseness or non erection of penis) and some scholars state that it is of five types namely Vātaja, Pittaja, Kaphaja, Raktaja and Sannipātaja.

Meḍhraśvayathu (swelling in the penis) and Meḍhra vedana (pain in the penis) etc. are the features of Vātaja; Sphoṭādi (formation of blisters etc.) are the features of Pittaja; Māṃsavṛddhi (abnormal muscle growth in the penis etc.) are the features of Kaphaja; Jvara (fever), Tṛṣṇā (thirst) etc., are the features of Raktaja and Agnineva dagdhasya Tīvrādāhaḥ (burning sensation of penis as if burnt with fire etc.) are the features of Sannipātaja.

Jarāja Klaibya Nidāna and Lakṣaṇa

◉ क्लैब्यं जरासम्भवं हि प्रवक्ष्याम्यथ तच्छृणु॥ जघन्यमध्यप्रवरं वयस्त्रिविधमुच्यते।
अतिप्रवयसां शुक्रं प्रायशः क्षीयते नृणाम्॥ रसादीनां सङ्क्षयाच्च तथैवावृष्यसेवनात्।
बलवीर्येन्द्रियाणां च क्रमेणैव परिक्षयात्॥ परिक्षयादायुषश्चाप्यनाहाराच्छ्रमात् क्लमात्।
जरासम्भवजं क्लैब्यमित्येतैर्हेतुभिर्नृणाम्॥ जायते तेन सोऽत्यर्थं क्षीणधातुः सुदुर्बलः।
विवर्णो दुर्बलो दीनः क्षिप्रं व्याधिमथाश्नुते ॥ एतज्जरासम्भवं हि ...। (Ca.Ci.30/176-181)

The age is classified into three types namely Jaghanya (early - childhood), Madhya (middle - adulthood) and Pravara (elder). Due to Rasādīnāṃ saṅkṣayāt (deficiency of the seven tissues), Avṛṣya sevanāt (not using aphrodisiacs), Balavīryendriyānāṃ krameṇa parikṣayāt (gradual loss of strength, energy, motor and sensory organs), Parikṣayādāyuṣa (decrease in longevity) Apyanāhārā (lack of food intake), Śramāt (physical exertion) and Klamāt (fatigue) lead to Śukraṃ kṣiyate (decreased semen) in Atipravayasāṃ (in old age). Subsequently, there will be Atyartha kṣīṇadhātu (excessive depleted tissues), Durbala (weakness), Vivarṇa (loss of complexion) and Durbalo dīnaḥ kṣipraṃ vyādhimathāśnute (poor resistance to disease). This Klaibya is due to Jarā (senility).

Śukrakṣayaja Klaibya Nidāna and Lakṣaṇa

◉ ... चतुर्थं क्षयजं शृणु। अतीव चिन्तनाच्चैव शोकात्क्रोधाद्भयात्तथा॥
ईर्ष्योत्कण्ठामदोद्वेगान् सदा विशति यो नरः। कृशो वा सेवते रूक्षमन्नपानं तथौषधम्॥
दुर्बलप्रकृतिश्चैव निराहारो भवेद्यदि। असात्म्यभोजनाच्चापि हृदये यो व्यवस्थितः॥
रसः प्रधानधातुर्हि क्षीयेताशु ततो नृणाम्। रक्तादयश्च क्षीयन्ते धातवस्तस्य देहिनः॥
शुक्रावसानास्तेभ्योऽपि शुक्रं धाम परं मतम्। चेतसो वाऽतिहर्षेण व्यवायं सेवतेऽति यः॥
तस्याशु क्षीयते शुक्रं ततः प्राप्नोति सङ्क्षयम्। घोरं व्याधिमवाप्नोति मरणं वा स गच्छति॥
शुक्रं तस्माद्विशेषेण रक्ष्यमारोग्यमिच्छता। (Ca.Ci.30/181-186)

Atīvacintanā (excessive worries), Śoka (grief), Krodha (anger), Bhayāt (fear), Īrṣyā (jealous), Utkaṇṭhā (regretting), Mada (intoxication), Udvegān (anxiety), Kṛśo vā rūkṣamannapānaṃ auṣadha sevanāt (emaciated or intake of non-unctuous diet, drinks and drugs), Durbala prakṛti (weak by nature), Nirāhārā (fasting) and Asātmya bhojanāt (intake of unwholesome food substances), leads to Rasa dhātu kṣaya in Hṛdaya, which in turn causes Raktādi dhātu kṣaya in sequence till it affects the Śukra dhātu which is considered to be superior dhātu among Sapta dhātus. However, Cetaso vāatiharṣeṇa vyavāya sevati (if the person indulges in sexual intercourse due to excitation of mind) leads to Śukra kṣaya immediately, thus he gets emaciated resulting in Ghoraṃ vyādhimavāpnoti maraṇaṃ vā sa gacchati (manifestation of serious diseases or death). Hence the Śukra should be protected especially who desire Ārogya (health).

Ācārya Suśruta classified Klaibya into six types namely Mānasa, Saumyadhātu kṣayaja, Śukrakṣayaja, Puṃstvopaghātaja, Sahaja and Sthiraśukra nimittaja. Ācārya Bhāvaprakāśa classified Klaibya into seven types namely Mānasa, Pittaja, Śukrakṣayaja, Meḍhra rogaja, Vīryavāhisirācchedaja, Sthiraśukra nimittaja and Sahaja

Mānasika Klaibya

◉ तैस्तैर्भावैरहृद्यैस्तु रिरंसोर्मनसि क्षते ॥ द्वेष्यस्त्रीसम्प्रयोगाच्च क्लैब्यं तन्मानसं स्मृतम् । (Su.Ci.26/9-10)

◉ तैस्तैर्भावैरहृद्यैस्तु रिरंसोर्मनसि क्षते । ध्वजः पतत्यतो नृणां क्लैब्यं समुपजायते ।
द्वेष्यस्त्रीसम्प्रयोगाच्च क्लैब्यं तन्मानसं स्मृतम् । (B.P.U.1/3)

Due to Hṛdyastu riṃsormanasi kṣate (mind inflicted with some unpleasant thoughts), the sexual desire ceases and Dhvajaḥ patati (penis become flaccid) or Dveṣyastrīsamprayogā (intercourse with a disliked woman) leads to Mānasika Klaibya (psychological impotency).

Saumyadhātu Kṣayaja/Pittaja Klaibya

◉ कटुकाम्लोष्णलवणैरतिमात्रोपसेवितैः ॥ सौम्यधातुक्षयो दृष्टः क्लैब्यं तदपरं स्मृतम् । (Su.Ci.26/10-11)

◉ कटुकाम्लोष्णलवणैरतिमात्रोपसेवितैः ॥ पित्ताच्छुक्रक्षये दृष्टः क्लैब्यं तस्मात् प्रजायते । (B.P.U.1/4)

Due to Kaṭukāmloṣṇalavaṇaratimātropasevitaiḥ (excessive intake of pungent, sour, hot and saline substances) leads to Saumyadhātu kṣaya (diminution of demulcent tissues) or Pitta prakopa (vitiation of Pitta) leads to Śukra kṣaya which in turn produces Klaibya.

Śukra Kṣayaja / Dhvajabhaṅgaja Klaibya

◉ अतिव्यवायशीलो यो न च वाजीक्रियारतः ॥ ध्वजभङ्गमवाप्नोति तच्छुक्रक्षयहेतुकम् । (Su.Ci.26/11-12)

◉ अतिव्यवायशीलो यो न च वाजीक्रियारतः ॥ ध्वजभङ्गमवाप्नोति स शुक्रक्षयहेतुकम् ॥ (B.P.U.1/5)

Ativyavāyaśīla yo na ca vājīkriyārataḥ (indulging in excessive sexual intercourse without using aphrodisiacs) leads to Śukra kṣaya which in turn produces Klaibya.

Puṃstvopaghātaja/Marmacchedaja Klaibya

◉ महता मेढ्ररोगेण मर्मच्छेदेन वा पुनः ॥ क्लैब्यमेतच्चतुर्थं स्यान्नृणां पुंस्त्वोपघातजम् । (Su.Ci.26/12-13)

◉ महता मेढ्ररोगेण चतुर्थी क्लीबता भवते । वीर्यवाहिसिराच्छेदान्मेहानानुन्नतिर्भवेत् ॥ (B.P.U.1/6)

Mahatā meḍhrarogeṇa (serious diseases of the penis) or Marmacchedena (destruction of the concerned vital areas) and Vīryavāhisirācchedā (destruction of the vessels or nerves that supply to the penis) leads to Puṃstvopaghātaja or Vīryavāhisirācchedaja Klaibya.

Śukranimittaja Klaibya

◉ बलिनः क्षुब्धमनसो निरोधाद् ब्रह्मचर्यतः । षष्ठं क्लैब्यं मतं तत्तु खरशुक्रनिमित्तजम् ॥ (Su.Ci.26/14)

◉ बलिनः क्षुब्धमनसो निरोधाद् ब्रह्मचर्यतः । षष्ठं क्लैब्यं स्मृतं तत्तु शुक्रस्तम्भनिमित्तकम् ॥ (B.P.U.1/7)

Balinaḥ kṣubdamanaso nirodhād brahmacaryata (voluntary suppression of the sexual desire by a strong man observing perfect continence) produces Kharaśukra (hardness of the semen) leading to Śukranimittaja Klaibya.

Sahaja Klaibya

◉ जन्मप्रभृति यः क्लीबः क्लैब्यं तत् सहजं स्मृतम् ॥ (Su.Ci.26/13)

◉ जन्मप्रभृति यत्क्लैब्यं सहजं तद्धि सप्तमम् ॥ (B.P.U.1/8)

Janmaprabhṛti yaḥ klībaḥ (sexual incapacity since birth) leads to Sahaja Klaibya.

Napuṃsaka

In addition to these, various types of Napuṃsaka are mentioned in the classical texts. Ācārya Caraka, Vāgbhaṭa and Suśruta described that during the second month of pregnancy, if the Kalala (embryo) become Arbudākṛti (like a tumour), then the born child become Napuṃsaka which is due to improper sexual activity of the parents at the time of conception i.e. having coitus in the Sandhyostu i.e. junction between the Yugma and Ayugma (even and odd days) and it comes under the Janmabala pravṛta vyādhi. Napuṃsaka is defined as the one who cannot perform the reproductive function and behaves neither as a male nor a female.

◉ तत्र शुक्रबाहुल्यात् पुमान्, आर्तवबाहुल्यात् स्त्री, साम्यादुभयोर्नपुंसकमिति ॥ (Su.Śā.3/5)

Śukrabāhulyāt (predominance of Śukra) leads to formation of Pumān (male fetus), where as Ārtavabāhulyāt (predominance of Ārtava) leads to Strī (female fetus) and Ubayorbāhulyāt (predominance of Śukra and Ārtava) leads to Napuṃsaka.

◉ षाण्ढ्यं नपुंसकता। (Ca.Ca.Sū.26/102-103)

Ṣaṇḍhata is Napuṃsaka (impotent or hermaphrodite)

◉ नपुंसकमिति न पुमान् न च स्त्री। (Ḍa.Su.Sū.29/5-7)

◉ नपुंसकस्य वा स्त्रीपुंसमानाकाररूपं । (Ca.Ca.Śā.4/14)

◉ उभयलिङ्गसङ्करे मिश्रीभावे नपुंसको विज्ञेयः । (In.A.S.Śā.2/34)

Those having mixed characters of Strī (female) and Puṃsa (male) are said as Napuṃsaka. And they are neither a male nor a female.

Napuṃsaka Bheda

◉ कस्माद्द्विरेताः पवनेन्द्रियो वा संस्कारवाही नरनारिषण्डौ। वक्री तथेर्ष्याभिरतिः कथं वा सञ्जायते वातिकषण्डको वा॥ (Ca.Śā.2/17)

Ācārya Caraka and Vāgbhaṭa described eight types of Napuṃsaka and Ācārya Suśruta described five types of Napuṃsaka.

Napuṃsaka	Caraka	Suśruta	Vāgbhaṭa
Dviretā	+	–	+
Pavanendriya / Vātendriya	+	–	+
Saṃskāravāhī	+	–	+
Naraṣaṇḍa	+	–	–
Nāriṣaṇḍa	+	–	–
Vakrī / Vakradhvajā	+	–	+
Īrṣyārati	+	–	+
Vātika ṣaṇḍa	+	–	+
Āsekya	–	+	+
Saugandhika	–	+	+
Kumbhīka	–	+	–
Īrṣyaka	–	+	–
Ṣaṇḍha	–	+	–

Dviretā

◉ बीजात् समांशादुपतप्तबीजात् स्त्रीपुंसलिङ्गी भवति द्विरेताः। (Ca.Śā.2/18)

If both the Śukra and Ārtava are in equal proportion and is afflicted with Vāta, then the child possesses the characters of both Strī (female) and Puṃsa (male) resulting in Dviretā. Hence, it indicates Hermaphrodite.

Pavanendriya/Vātendriya:

- शुक्राशयं गर्भगतस्य हत्वा करोति वायुः पवनेन्द्रियत्वम्॥ (Ca.Śā.2/18)

Due to the affliction of Śukrāśaya by Vata during intrauterine life, the person ejaculates Pavana (Vāyu) instead of Śukra at the end of sexual intercourse. It represents Aspermia.

Saṃskāravāhī

- शुक्राशयद्वारविघट्टनेन संस्कारवाहं कुरुतेऽनिलश्च। (Ca.Śā.2/19)

Affliction of Śukrāśaya dvāra (opening or orifice of Śukrāśaya) by the aggravated Vāta during intrauterine life causes Saṃskāravāhi, in which the person is able to do sexual intercourse only after taking the Vājikaraṇa drugs etc. Ācārya Cakrapāṇi opines that the conditions like Āsekya, Saugandhika and Kumbhīka can be included under Saṃskāravāhi as they require some stimulation for indulging in sexual intercourse.

Āsekya

- पित्रोरत्यल्पबीजत्वादासेक्यः पुरुषो भवेत् । स शुक्रं प्राश्य लभते ध्वजोच्छ्रायमसंशयम् ॥ (Su.Śā.2/38)

Due to Alpabījatvāt (scanty parental germ cells), the born child becomes a Āsekya, in which he gets erection undoubtedly after he swallows the semen of another man. This is also known as Mukhayoni because the man gets erection on tasting the semen discharged in his mouth during oral sex by another person.

Saugandhika

- यः पूतियोनौ जायेत स सौगन्धिकसञ्ज्ञितः । स योनिशेफसोर्गन्धमाघ्राय लभते बलम् ॥ (Su.Śā.2/39)

Due to Pūtiyoni (sordid vagina), the born child becomes Saugandhika, in which he gets Bala (strength or erection) after smelling the odour of Yoni (vagina) and Śepha (penis) of others. This is also known as Nāsāyoni.

Kumbhīka

- स्वे गुदेऽब्रह्मचर्याद्यः स्त्रीषु पुंवत् प्रवर्तते । कुम्भीकः स तु विज्ञेयः... । (Su.Śā.2/40)

In Kumbhīka, the person gets erection and indulges in coitus after becoming a passive member of an act of sodomy and then again commits sodomy with the female partner. This is also known as Gudayoni.

Narașaṇḍa and Nāriṣaṇḍa / Ṣaṇḍaka

- मन्दाल्पबीजावबलावहर्षौ क्लीबौ च हेतुर्विकृतिद्वयस्य॥ (Ca.Śā.2/19)

When the man and woman have Mandālpabījā (weak and deficient bīja) and are Abala (weak) and Aharṣa (not excited) during the sexual intercourse, then the born child becomes Narașaṇḍa and Nāriṣaṇḍa.

- .....षण्ढकं शृणु पञ्चमम् ॥ यो भार्यायामृतौ मोहादङ्गनेव प्रवर्तते । ततः स्त्रीचेष्टिताकारो जायते षण्ढसञ्ज्ञितः ॥ ऋतौ पुरुषवद्वाऽपि प्रवर्तेताङ्गना यदि । तत्र कन्या यदि भवेत् सा भवेन्नरचेष्टिता ॥ (Su.Śā.2/41-43)

Ācārya Suśruta says that when the person acts like a female during sexual intercourse with his wife due to Mohā (delusion), then the born male child becomes Narașaṇḍa and exhibits effeminate traits in his character (male inverts). If the female acts like a male during sexual intercourse due to Mohā (delusion), then the born female child becomes Nāriṣaṇḍa and exhibits masculine traits in her character (female inverts).

Īrṣyārati / Īrṣyaka

- ईर्ष्याभिभूतावपि मन्दहर्षावीर्ष्यारतेरेव वदन्ति हेतुम्॥ (Ca.Śā.2/20)
- ...ईर्ष्यकं शृणु चापरम् ॥ दृष्ट्वा व्यवायमन्येषां व्यवाये यः प्रवर्तते । ईर्ष्यकः स तु विज्ञेयः,... । (Su.Śā.2/40-41)

When the person gets stimulus for the sexual intercourse after seeing the sexual intercourse of another couple, then it is called Īrṣyārati. This child is born due to the sexual intercourse between male and female who are Īrṣyābhibhūta (envious to each other) and Mandaharṣa (have lack of sex-desire). It refers to scoptophilia or voyeurism.

Vakrī / Vakradhvajā

- मातुर्व्यवायप्रतिघेन वक्री स्याद्बीजदौर्बल्यतया पितुश्च। (Ca.Śā.2/20)
- यदा पुनरुभावपि भवतः स्त्रीपुंसौ तद्विधौ तदा वक्रध्वजो भवति। तस्य नैव ध्वजः स्तभ्यते ॥ (A.S.Śā. 2/40)

When the person develops Vakradvajā (penile curvature) associated with Naiva dhvajaḥ stambyate (lack of rigidity), it is called Vakrī.

This condition is produced in the offspring when sexual intercourse is done while the Māturvyavāyapratighane (mother is not having the desire or in abnormal posture) or when the normal position of the father and mother is reversed owing to the sexual weakness of the father and due to the Pituśca bīja daurbalyāt (poor quality of the sperms of the father).

Vātika Ṣaṇḍa

- वाय्वग्निदोषाद्वृषणौ तु यस्य नाशं गतौ वातिकषण्डकः सः। इत्येवमष्टौ विकृतिप्रकाराः कर्मात्मकानामुपलक्षणीयाः॥ (Ca.Śā.2/21)

The aggravated Vāta and Agni destroying the Vṛṣaṇa (testicles) during intrauterine life lead to Vātika ṣaṇḍa.

- आसेक्यश्च सुगन्धी च कुम्भीकश्चेर्ष्यकस्तथा । सरेतसस्त्वमी ज्ञेया अशुक्रः षण्ढसञ्ज्ञितः ॥ अनया विप्रकृत्या तु तेषां शुक्रवहाः सिराः । हर्षात् स्फुटत्वमायान्ति ध्वजोच्छ्रायस्ततो भवेत् ॥ (Su.Śā.2/44-45)

Finally, Ācārya Suśruta concluded that the Āsekya, Saugandhika, Kumbhīka and Īrṣyaka are having Śukra but they require some sort of stimulation for erection, where as the Ṣaṇḍa is completely devoid of Śukra. By the above described perversions, their Śukravahā sirā gets engorged in Āsekya etc., leading to erection of penis.

Sādhyāsādhyata of Klaibya

- ◉ केचित् क्लैब्ये त्वसाध्ये द्वे ध्वजभङ्गक्षयोद्भवे। वदन्ति शेफसश्छेदाद्वृषणोत्पाटनेन च॥
  मातापित्रोर्बीजदोषादशुभैश्चाकृतात्मनः। गर्भस्थस्य यदा दोषाः प्राप्य रेतोवहाःसिराः॥
  शोषयन्त्याशु तन्नाशाद्रेतश्चाप्युपहन्यते। तत्र सम्पूर्णसर्वाङ्गः स भवत्यपुमान् पुमान्॥
  एते त्वसाध्या व्याख्याताः सन्निपातसमुच्छ्रयात्। (Ca.Ci.30/188-191)

According to some scholars, Dhvajabhaṅgaja and Kṣayaja Klaibya are Asādhya (incurable). In this context, Dhvajabhaṅga caused by Śephasaśchedādvṛṣaṇotpāṭanena (excision of the phallus and testicles) is considered to be Asādhya (incurable) and Kṣayaja refers to condition which occurs due to Mātapitrorbījadoṣād (genetic defects of the parents) and Śubhaiścākṛtātmanaḥ (sinful actions of the past life); during the Garbhasthasya (intrauterine stage of foetus) the doṣās afflicts the Retovahā sirās and does Śoṣayantyāśu (desiccate the channels quickly) resulting in Retonāśā (elimination of semen) leading to Apumān (impotent man) even after Saṃpūrṇasarvāṅga (development of complete organs). These are said to be Asādhya (incurable) due to aggravation of three doṣas simultaneously.

- ◉ असाध्यं सहजं क्लैब्यं मर्मच्छेदाच्च यद्भवेत् । साध्यानामितरेषां तु कार्यो हेतुविपर्ययः ॥ (Su.Ci.26/15)

Sahaja Klaibya and Marmacchedaja Klaibya are said to be Asādhya (incurable). While the other types are Sādhya (curable) and they should be treated by Hetuviparyaya kārya (measures opposite to causative factors).

In Contemporary Science,

Klaibya can be correlated with erectile dysfunction in modern science. (Please refer to Topic 49)

VANDHYATVA

Introduction

The term Vandhya originates from the root word "Vandha" with the addition of the "Yak" Suffix (बन्ध + यक्), signifying barren, unfruitful, unproductive, fruitless, useless, defective, deprived, and destitute. Hence, it refers to a woman experiencing any form of impediment to the normal process of conception. Ācārya Suśruta explained Vandhyatva under the Vātaja Yoni roga, whereas Ācārya Caraka explained Vandhyatva as a Bījabhāga pradoṣāt, considered it as Asādhya Vyādhi (incurable disease). Ācārya Ḍalhaṇa has mentioned that while the term "Vandhya" is not commonly found in objective science, it is encompassed within the concept of Yonidoṣa. Ācārya Bhela expounded that seminal discharge into the Bāhya Nāḍi does not result in conception. Similarly, Ācārya Bhāvaprakāśa explained that Śukra falling on the Samīraṇa Nāḍi does not culminate in pregnancy. Ācārya Hārīta notably dedicated a separate chapter in the Tṛtīya sthāna to this subject.

Nirūkti and Paribhāṣa

- ◉ वन्ध्या निरपत्या स्त्री ॥ (Āyurvedīya Śabdakośa)

Vandhyā refers to a woman who is Nirapatyā (without offspring).

- ◉ वन्ध्यां नष्टार्तवां विद्यात् । (Su.U.38/10) / (M.N.62/2-4)
- ◉ वन्ध्या निरार्त्तवा ज्ञेया । (B.P.M.70/6)

Vandhyā refers to Naṣṭārtavāṃ (loss of menstruation) or Nirārtavā (absence of menstruation).

- ◉ वन्ध्यामाह– नष्टमार्तवं रजो यस्यां सा तथा। (Āt.M.N.62/2-4)

The term "Vandhyā" refers to a woman who experiences the loss of her menstrual cycle (Naṣṭārtavāṃ).

- ◉ यदा ह्यस्याः शोणिते गर्भाशयबीजभागः प्रदोषमापद्यते, तदा वन्ध्यां जनयति; । (Ca.Śā.4/30)
- ◉ यदा ह्यस्याः शोणिताख्ये बीजे गर्भाशयस्थे निर्वर्तकं बीजं प्रदुष्यति तदा वन्ध्यां जनयति॥ (A.H.Śā.2/45)

When the bījabhāga (part of the ovum) of Śoṇita, which is responsible for the Garbhāśaya (uterus), becomes vitiated, it results in Vandhyatva.

Nidāna and Saṃprāpti

- ◉ ध्रुवं चतुर्णां सान्निध्याद्गर्भः स्याद्विधिपूर्वकम् । ऋतुक्षेत्राम्बुबीजानां सामग्र्यादङ्कुरो यथा ॥ (Su.Śā.2/33)
- ◉ ऋतुरङ्गनाया रजःसमयः, क्षेत्रं गर्भाशयः, अम्बु पुनराहारपाकजो व्यापी रसधातुः, बीजं स्त्रीपुंसयोरार्तवशुक्रे। (Ḍa. Su.Śā.2/33)

Ācārya Suśruta explained the following four factors are responsible for Garbha (conception).

1. **Ṛtu** refers to the ovulation period (Rajaḥ samayaḥ).
2. **Kṣetra** refers to the uterus (Garbhāśaya).
3. **Ambu** refers to the nutrient fluid derived from sustenance (Rasadhātu obtained from Āhāra Rasa).
4. **Bīja** refers to Ārtava and Śukra.

The failure of any of these factors may result in Vandhyatva.

- ◉ मातृतः पितृत आत्मतः सात्म्यतो रसतः सत्त्वत इत्येतेभ्यो भावेभ्यः समुदितेभ्यो गर्भः सम्भवति। तस्य ये येऽवयवा यतो यतः सम्भवतः सम्भवन्ति तान् विभज्य मातृजादीनवयवान् पृथक् पृथगुक्तमग्रे॥ (Ca.Śa.4/4)

The Garbha (embryo) is formed from the combination of factors from the Mātṛtaḥ (mother), Pitṛtaḥ (father), Ātmataḥ (soul), Sātmyataḥ (wholesomeness), Rasataḥ (nutritive fluid), and Sattvataḥ (the mind). Abnormality of any one of these Ṣaḍbhāvas (six factors) results in Vandhyatva (infertility).

- मिथ्याचारेण ताः स्त्रीणां प्रदुष्टेनार्तवेन च। जायन्ते बीजदोषाच्च दैवाच्च शृणु ताः ....॥ (Ca.Ci.30/8)

Yoni rogas are observed in women as a result of their Mithyācāreṇa (faulty mode of life and diet), Praduṣṭenārtavena (vitiated menstrual blood), Bīja doṣāt (defects in ovum) and Daivācca (effects of past deeds).

- योनिप्रदोषान्मनसोऽभितापाच्छुक्रासृगाहारविहारदोषात्। अकालयोगाद्बलसङ्क्षयाच्च गर्भं चिराद्विन्दति सप्रजाऽपि॥ (Ca.Śā.2/7)

Due to Yoni pradoṣāt (defects in the genitals), Manasoabhitāpāt (psychological perturbation), Śukrāṣṛg doṣāt (defects of sperm and ovum), Āhāravihāra doṣāt (faulty diet and mode of life), Akālayogāt (coitus during improper period) and Balasaṅkṣayāt (diminution of strength), the women conceives with delay in spite of being fertile.

Due to the aforementioned Nidāna, there is an aggravation of Vāta doṣa, along with Pitta and Kapha doṣa, resulting in Bīja doṣa duṣṭi. This subsequently leads to Ārtava vaha Srotoduṣṭi and Naṣṭārtavā, culminating in the condition known as Vandhyatva.

Samprāpti Ghaṭaka of Vandhyā

Doṣa	Vāta Pradhāna Tridoṣas
Dūṣya	Rasa and Rakta dhātu
Agni	Jaṭharāgni Dhātvāgnimāndhya
Āma	Jaṭharāgni Dhātvāgnimāndhya janya Āma
Srotas	Ārtava vaha and Śukravaha
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Ārtavavaha and Śukravaha Srotas
Adhiṣṭhāna	Trayāvartā Yonimārga, Vṛṣaṇa and Meḍhra
Udbhava sthāna	Pakvāśaya
Rogamārga	Abhyantara

Vandhyā Bheda

Ācārya Caraka classified Vandhyā in to three types, namely

1. **Vandhyā** connotes a state of complete incapacity to conceive, often arising from inherent issues such as Bījopaghāta.
2. **Apraja** refers to the condition of Vandhyatva, wherein a woman achieves conception through therapeutic means, or experiences unsuccessful pregnancies despite achieving conception.
3. **Sapraja** pertains to a state in which a woman in her productive reproductive phase does not achieve conception, despite having a history of successful pregnancies.

- मृतवन्ध्या जन्मवन्ध्या काकवन्ध्या तु मोचिका ॥ गर्भस्रावीति मीयन्ते दोषैरेतैर्युतः स्त्रियः ॥ (Basavarājīyam. 15/4-5)

In Basavarājīyam, Vandhyā is classified in to four types, namely

1. **Mṛta Vandhyā :** If the baby dies after delivery, then the mother is called Mṛta Vandhyā.
2. **Janma Vandhyā :** If a woman has not yet conceived.
3. **Kākā Vandhyā :** If a mother has not conceived after two deliveries
4. **Mocikā (Kadalī Vandhyā) :** If a woman, after delivery, does not conceive for the second time.

- वन्ध्या स्यात्षट्प्रकारेण बाल्येनाप्यथवा पुनः । गर्भकोशस्य भङ्गाद्वा तथा धातुक्षयादपि ॥ जायते न च गर्भस्य सम्भूतिश्च कदाचन । काकवन्ध्या भवेच्चैका अनपत्या द्वितीयका ॥ गर्भस्रावी तृतीयाऽथ कथिता मुनिसत्तमैः । मृतवत्सा चतुर्थी स्यात्पञ्चमी च बलक्षयात् । (Hā.Sa.Tṛ.48/1-4)

Ācārya Hārīta classified the disease Vandhyā into six types, namely.

1. **Kāka Vandhyā :** refers to the inability to conceive after the birth of one child.
2. **Anapatyā Vandhyā :** refers to the condition where a woman has never conceived.
3. **Garbhasrāvī Vandhyā :** refers to a series of unsuccessful pregnancies due to recurring abortions.
4. **Mṛtavatsā Vandhyā :** refers to recurrent intrauterine fetal demise, stillbirths, and perinatal losses.
5. **Balakṣayāt Vandhyā :** Vandhytva resulting from a depletion of Bala (strength)
6. **Bālya, Garbhakośa bhaṅgā and Dhātukṣayāt Vandhyā :** refers to Vandhayatva resulting from Bālya (childhood), Garbhakośa bhaṅgā (damage to the uterus) and Dhātukṣayāt (depletion of dhātus)

⦿ बन्ध्याङ्गनानां नवधा प्रभेदाः प्रकीर्तिताः प्राक्तनवैद्यवर्यैः । तत्रादिबन्ध्या प्रथमा तु बोध्या तदुद्भवः स्यादतिपापकर्मभिः ॥
रक्तेन वातेन तथैव पित्तेन श्लेष्मणा चापि समस्तदोषैः । एवं भवेत् पञ्चविधाऽथ तिस्रो भूतेन दैवेन तथाऽभिचाराद् ॥
तथा प्रकारान्तरतोऽपि बन्ध्या चतुर्विधा संकथिता पुनश्च । तत्रादिमा वै गदिता तु गर्भस्राविण्यथाऽन्या मृतपुत्रिका च ॥
कन्याप्रसूः स्याच्च मता तृतीया सकृत्प्रसूर्या नियतं चतुर्थी । एषैव लोके किल काकबन्ध्या निगद्यते वैद्यजनैरवश्यम् ॥ तथैव
बन्ध्या भवतीह नारी तथैव बन्ध्योऽपि पुमान् प्रकीर्त्तितः । तच्छुक्रमध्यस्थितदोषजातैः सर्वे सुधीभिः स्वधियैव बोध्यम् ॥
(M.N.Pariśiṣṭam.Vandhyāroganidānam)

Ācārya Mādhavakara classified the disease Vandhyā into nine types, namely

1. Ādi Vandhyā (due to misdeeds of past and present life)
2. Raktena (Raktaja)
3. Vātena (Vātaja)
4. Pittena (Pittaja)
5. Śleśmamā (Kaphaja)
6. Samastadoṣa (Sannipātaja)
7. Bhūtena (infections etc.)
8. Daivena (idiopathic factors)
9. Abhicārād (due to improper acts)

Again he classified Vandhyā into four types namely

1. Garbha srāvī (recurrent miscarriage)
2. Mṛtaputrikā (still birth)
3. Kanyāprasūḥ (has only female child)
4. Sakṛtprasūryā (has only one child)

In Contemporary Science,

Vandhyā can be correlated with infertility in contemporary science. (Please refer to Topic 49)

MCQ's

1. Klaibya means
   a) Ṣaṇḍata b) Maithunāśakti
   c) Strīṣvanustsāha d) None of the above
2. Klaibya Causes
   a) Dhvajānucchrāya b) Maithunāśakti
   c) Strīṣvanustsāha d) All of the above
3. According to Caraka, Klaibya is .... types
   a) 3 b) 4
   c) 2 d) 5
4. Nature of Srāva in Dhvajabhaṅga Klaibya
   a) Dadhisannibhā b) Majjāmiśrita
   c) Pulākodakasaṅkāśa d) Vāruṇisannibhā
5. Types of Dhvajabhaṅga Klaibya
   a) 3 b) 4
   c) 2 d) 5
6. According to Caraka and Vāgbhaṭa, Napuṃsaka is of .... types
   a) 8,8 b) 8,5
   c) 8,6 d) 8,4
7. Naraṣaṇḍa and Nāriṣaṇḍa are due to
   a) Mandabīja b) Alpabīja
   c) Aharṣa d) All of the above
8. Bījabhāga Pradoṣa leads to
   a) Vandhyā b) Pūtipraja
   c) Vārtā d) Tṛṇa Putrika

9. Śukrāśaya dvāra vighāṭṭana is the cause for
   - a) Dviretā
   - b) Pavanendriya
   - c) Vātika Ṣaṇḍa
   - d) Saṃskāravāhī

10. Doṣas involved in Vātika ṣaṇḍa
    - a) Vāyu and Agni
    - b) Vāyu and Ākāśa
    - c) Vāyu and Ambu
    - d) None of the above

11. According to Caraka, Vandhyā is ____ types
    - a) 3
    - b) 4
    - c) 5
    - d) 6

Answer Keys

1	a	2	d	3	b	4	c	5	d	6	a
7	d	8	a	9	d	10	a	11	a		

TOPIC 49

Sexual Dysfunction and Infertility

SEXUAL DYSFUNCTION

Introduction

Sexual dysfunction refers to difficulties experienced by individuals or partners during the sexual response cycle. This cycle consists of four stages, beginning with the excitement phase, characterised by initial arousal, desire for sex, and physical responses such as increased heart rate, blood flow, and lubrication. The plateau phase follows, marked by full arousal and increased sexual pleasure, preparing the body for orgasm. The orgasm phase is a sudden release of sexual tension, resulting in intense pleasure in the genitals, accompanied by ejaculation in males. Finally, the resolution phase is a calming down period after orgasm, where the body returns to its normal physiological state, leaving feelings of relaxation and satisfaction.

According to the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5), sexual dysfunction requires a person to feel extreme distress and interpersonal strain for a minimum of six months (excluding substance or medication-induced sexual dysfunction). This condition profoundly impacts an individual's quality of sexual life, affecting both men and women globally and the prevalence increases with age.

Sexual dysfunction encompasses not only physical disorders but also paraphilias, or disorders of sexual preference. Various factors contribute to sexual dysfunction, including physical issues like hormonal imbalances, chronic illnesses, and medications. Psychological factors, such as anxiety, depression, stress, relationship issues, and trauma, also play a significant role. Furthermore, lifestyle factors like substance abuse, smoking, and poor sleep habits can impact sexual function.

Classification of Sexual Dysfunction

Sexual dysfunction can be categorized into four main types:

- **Sexual desire disorders:** Also known as Hypoactive Sexual Desire Disorder (HSDD), Characterised by a persistent lack of sexual desire, resulting in distress. Symptoms vary between males and females, including decreased libido, reduced sexual thoughts, and avoidance of sexual activity.
- **Arousal disorders:** Involve difficulty or inability to respond to sexual stimuli, with symptoms including Erectile Dysfunction in males and lack of vaginal lubrication or genital sensation in females.
- **Orgasm disorders:** Characterised by delay or absence of orgasm despite adequate sexual stimulation, including delayed ejaculation, anorgasmia (inability to achieve orgasm) and reduced orgasmic intensity
- **Pain disorders:** Pain during intercourse that prevents sexual enjoyment, including: dyspareunia (painful intercourse), vaginismus (involuntary vaginal contractions), priapism (prolonged and painful erection) and painful ejaculation.

Sexual dysfunction in Males

Male sexual dysfunction encompasses a range of conditions that significantly impact sexual health, including Erectile Dysfunction (ED), and Premature Ejaculation (PE). These conditions are characterised by impairments in sexual functioning. The prevalence of male sexual dysfunction increases with advancing age, with studies indicating that 50-70% of men aged 40 to 70 experiences some degree of ED.

The risk factors associated with male sexual dysfunction include various physical conditions such as age, diabetes mellitus, cancer, stroke, hypertension, penile trauma, and neurological disorders, including multiple sclerosis and Parkinson's disease. Psychological influences, such as depression, anxiety, and stress, further contribute to this issue. Moreover, disturbances in central serotonin neurotransmission and compromised functioning of 5-HT postsynaptic receptors are considered relevant factors.

Furthermore, lifestyle choices exert a notable influence on sexual function. These choices encompass smoking, excessive alcohol consumption, the use of certain medications, such as antidepressants and antihypertensive, sleep disorders, relationship challenges, and low testosterone levels.

ERECTILE DYSFUNCTION

Introduction

Erectile Dysfunction (ED) is defined as the consistent or recurrent inability of a man to achieve and maintain an erection sufficient for sexual activity. Consistency is essential to this definition, and a minimum duration of three months is typically required for diagnosis. In some cases, such as trauma or surgically induced ED, a diagnosis may be assigned sooner. ED is not a normal part of aging, although it is often associated with various physiological and psychological changes related to age.

In the last two decades, awareness of ED as a prevalent issue has increased significantly. In 1995, an estimated 152 million men globally experienced ED, with projections suggesting this number may rise to approximately 322 million by 2025, particularly in developing countries like India. The prevalence of ED is less than 10% among men in their 20s, 5% to 17% in their 40s, and 15% to 34% in men in their 70s. The incidence of ED is higher in individuals with certain medical conditions, including diabetes mellitus, obesity, benign prostatic hyperplasia (BPH), cardiovascular diseases, and hypertension. There is a correlation between the severity of ED and cardiovascular disease, making ED a potential "sentinel symptom" for undiagnosed cardiovascular and peripheral vascular disease.

Smoking and specific medications for diabetes and cardiovascular diseases are significant risk factors for ED. Additionally, men who have undergone radiation or surgery for prostate cancer, as well as those with lower spinal cord injuries, demonstrates higher incidence rates. ED may be classified as Primary or Secondary, and further categorized as Organic or Psychogenic.

I. Primary and Secondary Erectile Dysfunction

- **Primary Erectile Dysfunction (ED):** Primary ED is defined as the inability to obtain or maintain an erection that is sufficient for satisfactory sexual intercourse from the onset of sexual activity.
- **Secondary Erectile Dysfunction (ED):** Secondary ED refers to individuals who were previously capable of achieving full erections but have experienced erectile dysfunction at some point in their lives.

II. Organic and Psychogenic Erectile Dysfunction

Organic Erectile Dysfunction:

Organic ED is characterised by the inability to obtain an erection that is firm enough for vaginal penetration or to sustain the erection until the completion of intercourse, caused by physical factors. The underlying causes of organic ED are categorized as follows:

- **Vasculogenic:** Vascular disease is the predominant cause of organic ED, as adequate blood supply is essential for the attainment of an erection. Conditions that impede blood flow contribute significantly to erectile failure. Common diseases affecting the arteries include atherosclerosis, Leriche syndrome (aortoiliac occlusive disease), and risk factors such as hypercholesterolemia, hypertension, pelvic and perineal trauma, pelvic irradiation, smoking, and diabetes mellitus.
- **Neurogenic:** Disorders affecting the sacral spinal cord or the autonomic nerves supplying the penis hinder the nervous system's ability to relax penile smooth muscle, resulting in ED. Neurogenic ED is frequently associated with conditions affecting the nervous system, particularly in patients with spinal cord pathologies or neuropathies, stroke, multiple sclerosis, Alzheimer's disease, Parkinson's disease, and epilepsy.
- **Endocrinologic:** While androgens are known to influence libido, their precise role in erectile function remains ambiguous. Individuals with castrate levels of testosterone are capable of achieving erections through visual or sexual stimuli; however, normal testosterone levels appear to be critical for erectile function, particularly in older males. Endocrine disorders are relatively uncommon but should be considered in cases of hypogonadism, hypothalamic or pituitary issues (including tumours), primary gonadal failure, poorly controlled diabetes, and hyperthyroidism.
- **Drug-induced:** Certain medications may disrupt the central neuroendocrine or local neurovascular control mechanisms of penile smooth muscle, thereby affecting the erectile process. These medications include anticholinergics, antidepressants, antihypertensives, barbiturates, estrogens, histamine H2 receptor antagonists, narcotics, and sympatholytics.
- **Mechanical:** Conditions such as Peyronie's disease, prolonged bicycle riding, and congenital or acquired abnormalities of the penis can contribute to impotence.

Psychogenic Erectile Dysfunction:

Psychogenic Erectile Dysfunction is primarily attributed to psychological factors such as performance anxiety, feelings of guilt, depression, and conflicts surrounding sexual issues. Additionally, experiences of sexual abuse during childhood and fears related to pregnancy or sexually transmitted infections may contribute. Individuals experiencing psychogenic impotence may exhibit complete erectile failure with one partner while achieving an adequate response with another. Furthermore, they may achieve an erection during self-stimulation but not during sexual intercourse. Two primary mechanisms contribute to the inhibition of erections in cases of psychogenic ED:

- Psychogenic stimuli targeting the sacral cord may inhibit reflexogenic responses, thereby blocking activation of vasodilator outflow to the penis.
- Excess sympathetic stimulation in an anxious individual may increase the tone of penile smooth muscle.

A Practical Evaluation of Men with ED

To differentiate between psychogenic and organic ED, the following distinctions are relevant:

Organic ED

- Progressive deterioration of erectile function
- Reduction in both morning and nocturnal erections
- Absence of erections during masturbation
- No alteration in libido
- Presence of co-morbid health conditions

In Psychogenic ED

- Typically presents in younger patients (under 40 years of age)
- Maintenance of morning and nocturnal erections
- Ability to achieve erections during masturbation
- May be specific to certain partners
- Often manifests with sudden onset

Diagnostic Evaluation of Men with ED

Sexual History

An exhaustive sexual history should encompass details regarding sexual orientation, previous and current relationships, current emotional state, onset and duration of erectile difficulties, and prior consultations and treatments. The sexual health status of the partner is also of significance. Comprehensive documentation regarding the rigidity and duration of both stimulated and morning erections, as well as issues related to sexual desire, arousal, ejaculation, and orgasm such as premature ejaculation, retarded ejaculation, painful intercourse, anorgasmia, and decreased libido should be recorded.

Physical examination

The physical examination must assess the genitourinary, endocrine, vascular, and neurological systems. It may uncover undiagnosed conditions such as Peyronie's disease (curvature of the penis due to fibrous scar tissue), genital lesions (either premalignant or malignant), prostatic enlargement, or symptoms indicative of hypogonadism (e.g., small testes, alterations in secondary sexual characteristics). A digital rectal examination is advisable, and cardiovascular evaluation is essential, which includes the measurement of blood pressure and heart rate, particularly if recent assessments have not been conducted in the prior 3 to 6 months.

Laboratory Investigations:

Recommended laboratory tests for men with ED typically include Blood glucose and lipid profile. Hormonal tests should include a morning sample of total testosterone, evaluations of thyroid function, along with tests for prolactin and luteinizing hormone. For men aged over 45, serum PSA (Prostate-specific antigen), serum acid phosphatase, and transrectal ultrasonography are valuable for ruling out prostate cancer.

Specialized diagnostic tests:

- **Nocturnal penile tumescence and rigidity test:** This test should be conducted over a minimum of two separate nights. A functional erectile mechanism is indicated by an erectile event achieving at least 60% rigidity, recorded at the tip of the penis, and lasting a minimum of 10 minutes.
- **Intracavernous injection test:** This diagnostic procedure supplies limited information regarding vascular status. A positive test is characterised by a rigid erectile response, wherein the penis cannot be bent, occurring within 10 minutes after the intracavernous injection and persisting for 30 minutes.
- **Duplex ultrasound of the penis:** This evaluation measures penile blood flow through colour duplex ultrasound in both flaccid and erect states. Normal results generally indicate a peak systolic blood flow exceeding 30 cm/sec, an end-diastolic velocity below 3 cm/sec, and a resistance index surpassing 0.85.
- **Biothesiometry:** This test employs vibration to assess the perception of sensation. A diminished perception of vibration may suggest nerve damage in the pelvic region.
- **Dynamic infusion cavernosometry or cavernosography:** These are standard method of diagnosing venous leakage should only be executed in patients who do not respond to oral therapies and are being considered for vascular reconstructive surgery.

EJACULATORY DYSFUNCTION

Introduction

Ejaculatory dysfunction is defined as the inability of a male to efficiently ejaculate semen from the penis at the moment of sexual climax. This condition constitutes the most prevalent form of male sexual dysfunction and is a common contributing factor to male infertility. Ejaculatory dysfunction is broadly classified into two categories.

- **Diminished ability to produce ejaculate,** including retrograde ejaculation, and anejaculation, typically arises from advanced age, underlying pathology, or iatrogenic causes.
- **Abnormal timing of ejaculation,** comprising premature ejaculation and delayed ejaculation, is more commonly linked to psychological factors.

Retrograde Ejaculation

Retrograde ejaculation, specifically, involves semen flowing back into the bladder instead of exiting through the penis. This condition can arise from various causes, including neuropathy associated with diabetes mellitus and certain medications such as alpha blockers (e.g., tamsulosin for benign prostatic hyperplasia or BPH). Additionally, surgical procedures for BPH, like transurethral resection of the prostate (TURP), can lead to permanent iatrogenic retrograde ejaculation.

Anejaculation

Anejaculation is characterised by the inability to ejaculate stemming from various causes such as seminal tract obstruction, neurological dysfunction, injury, radiation, or surgery. A notable example is radical prostatectomy for prostate cancer, which involves removing seminal vesicles, thereby eliminating semen production.

Delayed Ejaculation

Delayed ejaculation is marked by difficulty achieving ejaculation despite prolonged stimulation. This condition is often linked to psychological factors but can also be influenced by erectile dysfunction and certain medications, such as serotonin reuptake inhibitors.

Premature Ejaculation

Premature ejaculation is a prevalent sexual dysfunction defined by the occurrence of ejaculation earlier than desired, leading to significant distress for both partners. Research indicates that approximately 30 to 75 percent of men aged 18 to 59 experience this condition, with around 30 percent also presenting with erectile dysfunction.

The World Health Organization (WHO) characterizes premature ejaculation as the inability to delay ejaculation, resulting in ejaculation occurring either before or shortly after penetration or in the absence of sufficient erection.

Definitions from reputable organizations include:

- **International Society for Sexual Medicine:** Premature ejaculation can be categorized as ejaculation occurring within one minute of vaginal penetration, in cases of lifelong premature ejaculation, or as a clinically significant and bothersome reduction in latency time, often to approximately three minutes or less, in cases of acquired premature ejaculation. This condition is marked by an inability to delay ejaculation during all or nearly all instances of vaginal penetration, ultimately resulting in negative personal consequences that include distress, frustration, and avoidance of sexual intimacy.

- **The Diagnostic and Statistical Manual of Mental Disorders (DSM-5):** Premature ejaculation is characterised by ejaculation occurring within one minute of vaginal penetration, occurring 75 to 100 percent of the time, persisting for a minimum of six months, and causing significant distress. Furthermore, this condition is not attributable to nonsexual mental disorders, medical conditions, adverse drug effects, severe relational distress, or other notable stressors.

Premature ejaculation is predominantly a psychological issue, although its precise etiology remains largely unclear. Several factors contribute to this condition, which can be categorized as biological or psychological.

Biological factors include hormonal imbalances, including luteinizing hormone (LH), prolactin, and thyroid-stimulating hormone (TSH), inflammation or infection of the prostate or urethra, erectile dysfunction, genetic predisposition, hyperthyroidism, Neuropathies, such as multiple sclerosis, alcohol dependence, diabetes, and sleep deprivation. Psychological factors involve depression, anxiety, stress, guilt, unrealistic expectations about sexual performance, performance anxiety, negative self- image, relationship dynamics and feelings of self-loathing.

Classification

Premature ejaculation (PE) may be classified in several ways, including:

- **Primary or Lifelong Premature Ejaculation:** This type is evident from the individual's first sexual experience. It typically involves an intravaginal ejaculation latency time (IELT) of less than 1 minute in 80% to 90% of cases.
- **Secondary or Acquired Premature Ejaculation:** This condition develops after a period of normal ejaculatory function and generally presents with an IELT of less than 3 minutes.

PE can also be categorized as Global or Situational

- **Global PE:** This classification refers to instances where PE occurs consistently across all situations, with all partners, and under various forms of stimulation.
- **Situational PE:** This type is characterised by variability, depending on factors such as the partner, type of stimulation, situation, location, or other contextual elements.

PE can also be categorized as mild, moderate, or severe, based on the severity

- **Mild PE:** This occurs within 30 seconds to 1 minute following vaginal penetration.
- **Moderate PE:** This is defined as occurring within 15 seconds to 1 minute after vaginal penetration.

- **Severe PE:** This classification encompasses cases where ejaculation occurs prior to or during foreplay, at the initiation of sexual activity, or within approximately 15 seconds of vaginal penetration.

Other Classifications

- **Variable PE:** This is characterised by natural fluctuations in ejaculation time.
- **Premature-like ejaculatory dysfunction:** In this condition, individuals encounter difficulty in controlling ejaculation while maintaining a normal IELT. It is often accompanied by excessive concern or preoccupation regarding ejaculatory control.

Diagnosis

The diagnosis of premature ejaculation involves a comprehensive evaluation that includes a thorough medical history, physical examination, and psychological assessment.

The medical history encompasses an examination of sexual history, the onset, duration, and severity of PE, alongside existing medical conditions, medications, substance use, and psychological factors. The physical examination focuses on genital and neurological assessments, as well as the measurement of blood pressure and heart rate.

Diagnostic criteria include an Intravaginal Ejaculation Latency Time (IELT) of less than 1-2 minutes for lifelong PE or less than 3 minutes for acquired PE, frequency and severity of PE, and presence of distress, as outlined in the DSM-5. Additionally, a psychological evaluation involves administering questionnaires, such as the Beck Depression Inventory and the State-Trait Anxiety Inventory, along with conducting interviews with the patient and their partner to gain a comprehensive understanding of the condition.

Differential diagnosis of Male sexual dysfunction

The differential diagnosis of male sexual dysfunction encompasses various conditions affecting sexual desire, arousal, and orgasm. The primary differential diagnoses for ED would be hypogonadism, loss of libido, depression with low mood, and other psychological conditions. This condition may be the first manifestation of diabetes or cardiovascular disease, as well as depression. Differentiating between true erectile dysfunction and other sexual disorders, such as premature ejaculation, is essential and usually easily accomplished by obtaining a good sexual history of the patient.

Before diagnosing premature ejaculation, it is essential to consider various underlying conditions. A significant factor in this assessment is the presence of severely delayed orgasm in the female partner, as research indicates that the average duration for women to achieve climax ranges from 12 to 25 minutes. In instances of extreme delayed female orgasm, it is possible that nearly any male may be classified as experiencing premature ejaculation; thus, the sexual response of the female partner is a critical element in the evaluation process.

Furthermore, it is important to account for the potential adverse effects of psychotropic medications, as these can significantly affect sexual function. Conducting a thorough differential diagnosis is imperative for accurately identifying the underlying causes, thereby facilitating effective treatment and enhancing overall sexual health outcomes.

Sexual dysfunction in Females

Female sexual dysfunction includes various issues that disrupt the sexual response cycle, making it difficult to have a satisfying sexual experience. Common symptoms include loss of sexual desire, impaired arousal, inability to achieve orgasm and pain during sexual activities. Research indicates that Female Sexual Dysfunction reveals a widespread issue, affecting 25-63% of women globally. Prevalence rates vary across different age groups and regions. Studies show that 10-40% of women experience decreased sexual desire (HSDD), 5-20% suffers from female orgasmic disorder (FOD), and 7-18% report dyspareunia.

Age is a significant risk factor; with prevalence increasing from 21-31% among women aged 18-24 to 42-63% among those 65 and older. Other risk factors include menopause, medical conditions like diabetes and hypertension, medications such as antidepressants, lifestyle factors like smoking and excessive alcohol consumption, relationship issues, history of abuse or trauma, and cultural and societal factors.

Classification

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR) explains various categories of female sexual dysfunction, classified according to specific symptoms:

- **Genito-pelvic pain/penetration disorder:** This condition may be either lifelong or acquired. It encompasses the presence of at least one of the following symptoms for a duration of six months or more:
  - Deep pelvic pain and muscular tension experienced during penetration.
  - Superficial burning vulvovaginal pain triggered by minimal touch.
  - Fear or anxiety occurring before, during, or after penetration, which often results in decreased sexual desire or avoidance of sexual activity.
  - Reflexive tightening of the vaginal muscles upon attempted penetration, which may render penetration difficult or impossible.
- **Female sexual interest/arousal disorder:** This disorder is characterised by the absence of, or a decrease in, at least three of the following symptoms persisting for six months or longer:
  - Interest in engaging in sexual activity.
  - Initiation of sexual activity and responsiveness to a partner's initiation.
  - Experience of excitement or pleasure during nearly all sexual activities.
  - Presence of sexual or erotic fantasies or thoughts.
  - Sensations, either genital or non-genital, during sexual activity.
  - Interest or arousal in response to internal or external sexual or erotic stimuli (e.g., written, verbal, visual).
- **Female orgasmic disorder:** This disorder is defined by the absence, significant reduction in intensity, or considerable delay of orgasm in response to sexual stimulation, despite high levels of subjective arousal. Symptoms must manifest during almost all sexual activities and must be present for at least six months. Acquired orgasmic disorders may be associated with the emergence of new conditions, including psychological or behavioural disorders, or anatomical changes (e.g., resulting from cancer or surgical interventions).
- **Substance/medication-induced sexual dysfunction:** This type of dysfunction is linked to the initiation, alteration in dosage, or cessation of a substance or medication.
- **Other specified and unspecified sexual dysfunction:** This category includes sexual dysfunction that does not meet to the criteria established for the aforementioned categories.

Diagnosis

Diagnosis of Female Sexual Dysfunction involves a comprehensive approach, considering physical, psychological, and relational factors. According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR), diagnostic criteria include:

- Persistent or recurrent lack of interest in sexual activity
- Difficulty achieving or maintaining arousal
- Difficulty achieving orgasm
- Pain during vaginal intercourse or genital stimulation

The diagnostic process comprises several critical components:

- **Medical history:** A thorough medical history is essential, including a detailed account of sexual history, the onset, duration, and severity of symptoms, existing medical conditions, medications, substance use, and psychological factors.
- **Physical examination:** The physical assessment includes a genital examination, which covers external inspection, vaginal examination (assessing factors such as pH, moisture, and elasticity), and cervical evaluation. A neurological examination assesses sensory function (touch, pressure, temperature, and vibration), motor function, and the bulbocavernosus reflex (BCR). Measurement of vital signs, including blood pressure and heart rate, is also conducted.

- **Psychological evaluation:** This may involve the administration of standardized questionnaires, such as the Female Sexual Function Index (FSFI) and the Female Sexual Distress Scale (FSDS).
- **Laboratory tests:** Relevant laboratory assessments may include hormone level evaluations, thyroid function tests, and other pertinent diagnostic tests as indicated.

Differential diagnosis of Female sexual dysfunction

Female sexual dysfunction encompasses various conditions affecting sexual desire, arousal, and orgasm. Hypoactive Sexual Desire Disorder (HSDD) may arise from several contributing factors, including psychological elements such as depression, anxiety, or interpersonal relationship difficulties. Additional causes may include hormonal imbalances encountered during menopause or related to thyroid dysfunction, medical conditions characterized by chronic illness or pain, side effects from certain medications, particularly antidepressants, and lifestyle influences such as stress, tobacco use, and excessive alcohol consumption.

Female Sexual Arousal Disorder (FSAD) can result from vulvodynia, vaginismus, hormonal imbalances, neurologic disorders like multiple sclerosis or spinal cord injury, or medications. Female Orgasmic Disorder may be caused by psychological factors, hormonal imbalances, neurologic disorders, medications, or pelvic surgery/trauma.

Dyspareunia, or painful intercourse, can be attributed to vulvodynia, vaginismus, endometriosis, pelvic inflammatory disease (PID), or sexual trauma/abuse. Other female sexual dysfunctions include female genital arousal disorder, persistent genital arousal disorder, and sexual aversion disorder.

INFERTILITY

Introduction

Infertility is a medical condition affecting the male or female reproductive system, characterised by the inability to achieve pregnancy after 12 months or more of regular, unprotected sexual intercourse. It is estimated that approximately 180 million couples globally experience infertility, with around 25-28% (15-20 million) residing in India. A significant multinational study conducted by the World Health Organization (WHO) revealed that female infertility was identified as the sole cause in 37% of infertile couples. In 35% of cases, both male and female factors contributed to infertility, underscoring the complexity of reproductive health issues. Furthermore, male factor infertility was responsible for 8% of cases, while 20% of cases remained classified as unspecified or unexplained.

Infertility categorizes into two types:

- **Primary infertility:** The inability to achieve any pregnancy,

- **Secondary infertility:** The inability to achieve pregnancy following a previous successful conception.

The underlying causes of infertility may be ascribed to male factors, including low sperm count, poor sperm motility, abnormal sperm morphology, varicocele, and hormonal imbalances. Female factors encompass ovulation disorders, tubal damage or blockage, endometriosis, polycystic ovary syndrome (PCOS), and hormonal imbalances. Additionally, lifestyle and environmental factors such as age, smoking, alcohol consumption, obesity, stress, and exposure to environmental toxins significantly contribute to infertility.

Male Infertility

Male infertility is defined by the World Health Organization (WHO) as the inability of a male to make to achieve pregnancy in a fertile female after a minimum of one year of regular unprotected intercourse. The causes of male infertility can be classified into three primary categories:

- **Pre-testicular causes:** These factors include hormonal imbalances or regulatory issues that influence testicular function. Conditions may encompass hypogonadotropic hypogonadism, erectile dysfunction, coital disorders, genetic factors, and chromosomal abnormalities.
- **Testicular causes:** This category pertains to primary defects within the testes that adversely affect sperm production (spermatogenesis), originating from conditions other than obstruction or dysfunction of the hypothalamic-pituitary-gonadal axis. These causes can be further subdivided into congenital, acquired, or idiopathic testicular failures
    - **Congenital testicular failure:** This may present as bilateral anorchia, testicular dysgenesis (including cryptorchidism), and genetic abnormalities such as Klinefelter's syndrome or Noonan's syndrome (male Turner syndrome).
    - **Acquired testicular failure:** This may result from trauma, testicular torsion, orchitis, exposure to gonadotoxins (including certain medications and radiation), systemic diseases (such as renal failure, hepatic disease, and sickle cell disease), varicocele, or surgical interventions that damage the vascular structures of the testes.
    - **Idiopathic testicular failure:** This category accounts for 25-50% of male infertility cases where a comprehensive evaluation does not reveal an identifiable underlying cause.
- **Post-testicular causes:** These factors encompass obstructions or issues that impede sperm transport or delivery. Examples include ejaculatory dysfunction and blockages in the epididymis, vas deferens, or ejaculatory duct. Such obstructions may be acquired (e.g., resulting from orchioplexy or vasectomy during hernia repair) or congenital (e.g., absence or atresia of the male ductal system).

Clinical Evaluation of Male Infertility

The American Society for Reproductive Medicine (ASRM) recommends that male infertility evaluation be initiated after one year of unprotected intercourse, as it is observed that 90% of planned pregnancies occur within this period. Nonetheless, initial screening is advisable upon the first presentation of infertility concerns, since prolonged subfertility may diminish the likelihood of effective treatment. It is important to note that couples frequently experience significant anxiety and stress after only a few months of unsuccessful attempts at conception, influenced by both internal and societal pressures.

Timely evaluation is paramount, and the initial assessment should be both rapid and non-invasive, while also being cost-effective. The evaluation typically focuses on medical history, physical examination, assessment of sperm quantity and quality, biochemical characterization of the ejaculate, and fundamental andrological tests.

History and Physical Examination:

The history should emphasize the identification of factors potentially related to impairment of reproductive function, commonly referred to as risk factors. These factors are elaborated upon in the subsequent sections:

- **Infertility History:** This section should encompass details regarding the age of both partners, the duration of their marriage, the length of time they have engaged in unprotected intercourse, prior contraceptive methods utilized, previous pregnancies, and any past or ongoing treatments for infertility.
- **Sexual History:** This should include information on the frequency of intercourse, the adequacy of penile penetration, details regarding ejaculatory function, and orgasmic sensation. Additionally, attention should be directed towards the understahding of the fertility period by the individuals involved.
- **Childhood and Developmental History:** Information concerning any past illnesses that may affect fertility, such as prepubertal mumps orchitis, undescended testicles, testicular torsion, and testicular trauma, should be documented.
- **Medical History:** Certain hereditary conditions may impact fertility, including diabetes mellitus, thyroid disorders, tuberculosis, and vascular diseases. Such conditions must be recorded diligently. Moreover, any febrile incidents occurring within the preceding six months may lead to a temporary reduction in sperm quantity and quality. It is also imperative to investigate the presence of sexually transmitted infections (STIs) such as syphilis, gonorrhea, mycoplasma, and non-specific urethritis, as these may adversely affect sperm function.
- **Surgical History:** Any surgical interventions possibly associated with infertility should be recorded. This includes procedures related to hypospadias, urethral stricture, bladder neck operations, vasectomy, varicocele, inguinal hernia, testicular torsion, sympathectomy, hydrocele, and prostatectomy.

- **History of Drugs and exposure to Chemicals:** Certain pharmacological agents and substances can be detrimental to the male reproductive system. Chemotherapeutic agents, cimetidine, nitrofurantoin, marijuana, and anabolic steroids have demonstrated adverse effects. Moreover, the consumption of alcohol, tobacco, and narcotics, as well as regular use of hot baths or saunas, may contribute to temporary infertility.
- **Review of Systems:** Specific respiratory conditions, such as childhood bronchiectasis, have been associated with azoospermia due to obstruction.

Physical examination:

This should comprise both a general examination and a urogenital evaluation. Any factors that may affect overall health can theoretically influence sperm production; therefore, the physical examination must be comprehensive, with significant emphasis placed on the genitalia.

General Examination:

The general examination assesses overall health and should include the following

- Evaluation of body habitus, hair distribution, gynecomastia, and fat distribution to identify potential disorders such as Cushing's syndrome, hypogonadism, or hypothyroidism.
- Assessment of height, weight, blood pressure, extremity length, and nutritional status.
- Anosmia testing to exclude Kallmann syndrome in patients with hypogonadism.
- Evaluation for hyperprolactinemia to rule out space-occupying lesions within the fossa turcica

Urogenital Examination:

The urogenital examination focuses on the genitalia and should be conducted in a warm environment to promote relaxation of the scrotal dartos muscle. This examination includes:

- Inspection of the penis, urethral opening, and prepuce for any abnormalities.
- Palpation of the scrotal contents to evaluate testicular localization, consistency, and tenderness.
- Measurement of testicular size utilizing an orchidometer, noting that volumes less than 15 ml are often correlated with abnormal spermiograms.
- Evaluation of the epididymis and vas deferens for cysts, tenderness, or thickening, indicative of prior or chronic inflammation.

- Assessment for scrotal swelling, including inguinal hernias and hydroceles, varicocele, or spermatic venous reflux.
- Examination of the groin for lymphadenopathy, scars, or other surgical evidence.
- Digital rectal examination of the prostate and seminal vesicles for comprehensive assessment.

Endocrine Evaluation

Endocrine evaluation plays a critical role in identifying underlying hormonal imbalances that may contribute to abnormalities in semen. Hormonal testing should encompass the measurement of serum Follicle Stimulating Hormone (FSH), Luteinizing Hormone (LH), and Testosterone (T) levels.

The measurement of testosterone levels is indicative of Leydig cell function and proves particularly beneficial in cases presenting with delayed puberty, decreased libido, or impotence. The normal range for serum testosterone levels in adult males is between 241 and 827 ng/dl. Low testosterone levels, when accompanied by low or normal LH and FSH levels, suggest disturbances in gonadotrophin secretion.

Gonadotrophins, specifically LH and FSH, are responsible for regulating testicular functions. Elevated levels of these hormones indicate primary testicular dysfunction. Elevated FSH levels suggest seminiferous tubule dysfunction, while high FSH with normal LH levels indicates preserved Leydig cell function. A deficiency in gonadotrophins may indicate pituitary or hypothalamic damage.

Estrogen determination is necessary in cases of gynecomastia, significant obesity, or suspected end-organ resistance to testosterone. The normal ranges for serum FSH and LH levels are from 1.0 to 14.0 mIU/ml and from 0.7 to 7.4 mIU/ml, respectively.

Interpretation of the Hormonal Evaluation

Testosterone	LH	FSH	PRL	Cause
Low	High	High	Normal	Testicular failure
Normal	Normal	High	Normal	Isolated germinal failure
Low	Low	Low	High	Hypogaonadotrophic hypogonadism
Normal	Normal	Normal	Normal	Non-endocrine cause obstructive
High	High	Normal	Normal	Partial androgen resistance
Normal	Normal	Normal	High	Hyperprolactinaemia not the cause of Oligo / Azoospermia

Semen Analysis

Semen analysis is conducted to assess spermatozoa and seminal fluid parameters, thereby estimating overall semen quality. A conventional analysis includes the quantification of sperm concentration, motility, morphology, and seminal plasma, alongside non-spermatozoidal cell quantification and antisperm antibody detection.

The collection of samples requires that individuals obtain a specimen through masturbation into a clean container, abstaining from sexual activity for a period of 48 hours to 7 days. The specimen must be protected from extreme temperatures (20°C–40°C) and should be evaluated immediately following liquefaction, ideally within one hour.

The initial macroscopic examination evaluates the sample's appearance, liquefaction, consistency, volume, pH, and color. A normal sample appears grey-opalescent and homogeneous, liquefies within 60 minutes at room temperature, and has a consistency that allows it to leave the collection needle in small drops.

The initial microscopic assessment provides estimates of motility and concentration, detects non-spermatozoidal cells, and identifies any agglutination of spermatozoa. Sperm concentration is deemed normal at ≥15 million cells/mL, while oligospermia is classified as below 15 million cells/mL. Azoospermia indicates the absence of sperm cells entirely.

Sperm motility is classified as normal at ≥40% for progressive motility (PR) and non-progressive motility (NP), while asthenospermia signifies reduced motility. Sperm morphology is considered normal when >4% of forms are normal, whereas teratospermia denotes abnormal morphology.

Standard values according to the 2010 WHO criteria include:

Semen Volume	1.5 mL (normal range:1.5–5 ml)
pH	> 7.2
Sperm concentration	15 million/mL (normal range:12–16 million/mL)
Total no. of spermatozoa	39 million/ejaculate (normal range:33–46 million)
Total Motility (PR+NP)	40% (normal range: 38-42%)
Progressive Motility (PR)	32% (normal range: 31-34%)
Morphology	4% normal forms (normal range: 3-4%)
Vitality (live spermatozoa)	58% (normal range: 55-63%)
Leukocytes (106/mL)	< 1 million/ml

Anti Sperm Antibody Test:

Testing for anti-sperm antibodies is indicated when semen analysis reveals abnormal clumping or agglutination, unexplained decreases in motility, or abnormalities in the post-coital test. Screening tests include the Immunobead method and the Mixed Antiglobulin

Reaction (MAR) test. The Immunobead test is designed to detect IgG, IgA, and IgM antibodies, with positivity defined as 25% or more motile spermatozoa exhibiting Immunobead binding. The MAR test detects IgG antibodies, with positivity defined as 50% or more motile spermatozoa possessing adherent particles. Conditions correlated with anti-sperm antibodies include genital tract infections, varicocele, cryptorchidism, testicular torsion, and autoimmune diseases.

Sperm Function Test:

If previous evaluations are normal, a sperm function test must be considered to assess the sperm's capability to penetrate the ovum. This evaluation includes capacitation assays, acrosome reactions, sperm penetration assays, hypo-osmotic swelling tests, and vitality staining.

Imaging tests:

Imaging tests are employed to investigate potential obstructions in the ejaculatory duct as well as the anatomical structures of the testis, epididymis, and spermatic cord. Transrectal ultrasound serves to evaluate ejaculatory duct obstruction, while scrotal ultrasound and color Doppler imaging assess the anatomy of the testis, epididymis, and spermatic cord. A vasogram is utilized to ascertain the patency of the ductal system.

Post-Coital Test:

The post-coital test evaluates the interaction between sperm and cervical mucus. This test is indicated in instances of semen hyperviscosity, variations in semen volume, with adequate sperm density, or unexplained infertility.

Testicular Biopsy:

A testicular biopsy allows for the differentiation between obstructive and non-obstructive testicular pathologies, thereby providing essential prognostic information to inform medical therapy.

Computer Assisted Semen Analysis (CASA)

Computer-assisted semen analysis (CASA) automates the assessment of semen parameters, yielding quantitative evaluations of sperm motility. Parameters measured include curvilinear velocity (VCL), straight-line velocity (VSL), linearity (LIN%), amplitude of lateral head displacement (ALH), and beat cross frequency (BCF).

Biochemical analysis

Biochemical analysis examines accessory gland function through various markers such as citric acid, zinc, and acid phosphatase for the prostate gland, fructose and prostaglandins for the seminal vesicles, as well as free L-carnitine, glycerophosphocholine, and alpha-glucosidase for the epididymis. A reduced secretory function of these glands is indicated by a decline in the overall output of these markers, potentially resulting from infections or other underlying factors. Fructose evaluation is particularly useful for diagnosing dysgenesis of the seminal vesicles and ejaculatory duct obstruction.

Urinalysis:

Urinalysis plays a vital role in the evaluation of male infertility. A thorough urine analysis may indicate significant pyuria, which could suggest the presence of urethritis or prostatitis. Additionally, a notable quantity of sperm may be detected, pointing to the possibility of retrograde ejaculation; this condition can be further assessed through immediate post-ejaculate urine analysis. Moreover, urinalysis can uncover undiagnosed renal disease or diabetes, thereby providing essential insights into underlying conditions that may affect fertility.

Differential diagnosis of Male infertility

Differential Diagnosis of Male Infertility involves various hormonal and genetic disorders. These include adult growth hormone deficiency, 5-alpha reductase deficiency, androgen receptor gene polymorphisms, and chromosomal abnormalities such as Y chromosome microdeletions (AZFa, AZFb, AZFc) and Klinefelter syndrome. Hypogonadism and hypogonadotropic hypogonadism, as well as luteinizing hormone deficiency, also contribute to male infertility.

Physical and structural abnormalities play a significant role, including bilateral testicular torsion, cryptorchidism, bilateral vasectomy, ejaculatory duct obstruction, varicoceles, and Peyronie's disease. Systemic and chronic illnesses, such as Cushing's disease, celiac disease, hemochromatosis, liver failure, HIV infections, and cystic fibrosis, also impact fertility.

Environmental and lifestyle factors, including substance use (cimetidine, spironolactone), chemotherapy (cyclophosphamide), radiation exposure, lead exposure, pesticide and chemical exposure, obesity, and opioid abuse, contribute to male infertility. Infectious and immunological factors, such as sexually transmitted diseases, brain damage from tumors or trauma, and Hodgkin lymphoma, also affect fertility.

Developmental disorders, including Kallmann syndrome and immotile cilia syndrome, as well as Down syndrome, are additional considerations. Other medical conditions, such as testicular cancers and trauma, estrogen excess, and FSH abnormalities, complete the differential diagnosis of male infertility.

Female Infertility

Female infertility is defined as the inability to conceive after 12 months of regular, unprotected sexual intercourse for women under the age of 35, or after 6 months for women aged 35 and older. The causes of female infertility are varied and can include a wide range of factors. They are

- **Ovulatory Disorders** manifest as irregular or absent ovulation, encompassing conditions such as oligo-ovulation (irregular ovulation) or anovulation (absence of ovulation). These disorders are significant contributors to infertility, as the lack of oocyte (egg) release precludes the possibility of fertilization and subsequent pregnancy. The World Health Organization categorizes ovulatory disorders into four primary classifications:
    - **Hypogonadotropic hypogonadal anovulation:** This classification includes conditions such as hypothalamic amenorrhea.
    - **Normogonadotropic normoestrogenic anovulation:** An illustrative instance of this category is polycystic ovarian syndrome (PCOS).
    - **Hypergonadotropic hypoestrogenic anovulation:** This category encompasses conditions such as premature ovarian failure.
    - **Hyperprolactinemic anovulation:** This type is frequently associated with conditions such as pituitary adenomas.
- **Tubal Infertility:** Occurs due to:
    - **Blocked Fallopian Tubes:** caused by pelvic inflammatory disease, previous surgical interventions, or tubal ligation.
    - **Tubal Scarring:** Resulting from infectious processes, endometriosis, or historical surgeries.
- **Uterine or Cervical Causes:** Contributing to infertility may include:
    - **Uterine Fibroids:** These noncancerous growths can interfere with implantation and foetal development.
    - **Uterine Anomalies:** Such as septate or bicornuate uterus.
    - **Cervical Issues:** Conditions such as stenosis or inadequate cervical mucus that may impede sperm entry.
- **Endometriosis** represents another significant factor, wherein tissue resembling the uterine lining develops outside the uterus, leading to adhesions, scarring, and inflammation.
- **Immune System Disorders** can also play a role in infertility, as autoimmune reactions may target reproductive tissues.
- **Hormonal Imbalances** including conditions such as hyperprolactinemia, thyroid disorders, or cortisol dysregulation, further complicate fertility challenges.

Clinical Evaluation of Female Infertility

The clinical evaluation of female infertility is a systematic process intended to identify the underlying causes of infertility.

Medical History:

The evaluation commences with a detailed medical history that encompasses several critical areas:

- **Reproductive History:** The reproductive history is assessed, including the duration of infertility, obstetrical history, menstrual history (such as molimina), previous pregnancies, miscarriages, contraceptive use, and any previous fertility treatments.
- **General Health History:** The general health history is evaluated, considering chronic illnesses like diabetes and thyroid disorders, surgeries such as tubal ligation and cesarean section, medications including hormonal treatments, social and lifestyle factors like smoking, alcohol use, illicit drug use, exercise, and diet, occupation
- **Sexual history:** The sexual history is also assessed, focusing on the frequency and timing of sexual intercourse, potential issues with the male partner (e.g., erectile dysfunction, ejaculation difficulties), and sexual orientation.
- **Family history: The family** history is obtained to screen for genetic predispositions, **history of venous thrombo**tic events, recurrent pregnancy loss, and infertility.

Physical Examination:

A comprehensive physical examination is a crucial component of evaluating female infertility. The physical exam is divided into two main categories:

- **General Examination:** The examination begins with vital signs and calculating body mass index (BMI) to assess overall health. The physical exam also includes a thorough thyroid evaluation, a breast exam to check for galactorrhoea (spontaneous milk production), and signs of androgen excess, such as acne, hirsutism, or external genitalia abnormalities.
- **Pelvic Examination:** The pelvic examination is also critical, focusing on identifying abnormal vaginal or cervical anatomy, pelvic masses or tenderness, uterine enlargement or irregularity, and assessing the reproductive organs for abnormalities. Specifically, the pelvic exam evaluates:
    - Uterine size and shape
    - Ovarian size and tenderness
    - Cervical abnormalities
    - Vaginal discharge or lesions

- **Transvaginal ultrasonography** is often performed at the bedside as part of the initial physical exam to provide a detailed visualization of the reproductive organs.

Laboratory Tests:

Initial laboratory assessments encompass the following components:

- **Hormonal Assays:** These evaluations measure the levels of essential hormones involved in regulating menstrual cycles, ovulation, and fertility. They are instrumental in identifying hormonal imbalances or disorders that may affect fertility.
    - **Follicle-Stimulating Hormone (FSH):** This test evaluates ovarian function and reserve.
    - **Luteinizing Hormone (LH):** This measurement assesses ovulation and the functionality of the pituitary gland.
    - **Estradiol (E2):** This test measures estrogen levels, which are critical for follicular development and ovulation.
    - **Prolactin:** This evaluation rules out hyperprolactinemia, a condition that can disrupt ovulation.
    - **Thyroid Hormones (TSH, free T4):** These measurements identify thyroid disorders that may impact fertility.
- **Ovulation Testing:** These assessments confirm the occurrence of ovulation and assist in identifying potential issues within the menstrual cycle..
    - **Basal Body Temperature (BBT) Charts:** These charts track temperature fluctuations throughout the menstrual cycle to detect ovulation.
    - **Ovulation Predictor Kits (OPKs):** These kits detect the LH surge, indicating the onset of ovulation.
    - **Serum Progesterone Levels:** This test measures progesterone levels to confirm that ovulation has taken place.

Based on the results of the initial assessments, additional tests may be recommended

- **Anti-Müllerian Hormone (AMH) Test:** This test evaluates ovarian reserve.
- **Inhibin B Test:** This assessment evaluates ovarian function and follicular development.
- **Androgen Levels (Testosterone, DHEA-S):** These tests identify excess androgens or conditions such as polycystic ovary syndrome (PCOS).
- **Insulin Resistance Tests (Glucose Tolerance Test):** These tests diagnose insulin resistance, a condition commonly associated with PCOS.

Imaging Studies:

Imaging studies play a crucial role in evaluating female infertility.

- **Ultrasound** technology utilizes high-frequency sound waves to produce images of reproductive organs, assessing ovarian size and function, uterine size and shape, and fallopian tube patency.
- **Hysterosalpingography (HSG),** an X-ray test using contrast dye, visualizes the reproductive tract to identify blockages or abnormalities in the fallopian tubes and uterine cavity, diagnosing tubal damage, adhesions, or endometriosis.

Specialized Tests:

- **Laparoscopy,** a minimally invasive surgical procedure, allows direct examination of pelvic organs, diagnosing endometriosis, adhesions, tubal damage, or ovarian cysts, with the option for simultaneous treatment.
- **Sonohysterography** (SHG), an ultrasound test using saline solution to expand the uterine cavity, evaluates the uterine cavity and detects abnormalities such as polyps or fibroids.

Genetic Testing:

Genetic testing identifies potential underlying genetic factors.

- **Karyotyping,** analyzes chromosomal structure to detect abnormalities affecting fertility and diagnose genetic disorders like Turner syndrome.
- **Genetic Screening** tests for specific conditions, including Fragile X syndrome, which impacts ovarian function and fertility, and other disorders like cystic fibrosis and sickle cell disease

Additional Tests:

- **Endometrial Biopsy** obtains a tissue sample from the uterine lining to evaluate endometrial thickness and receptivity, diagnosing endometrial disorders such as endometriosis or cancer.

Differential diagnosis of Female infertility

Female infertility is a complex disorder with profound effects on couples, often involving multiple underlying causes. Polycystic Ovary Syndrome (PCOS) is a prevalent condition among infertile women, necessitating thorough evaluation.

The differential diagnosis for PCOS includes several conditions that must be ruled out, such as androgen-producing ovarian tumors, adrenal tumors, nonclassic congenital adrenal hyperplasia (CAH), Cushing syndrome, prolactinemia disorders, and thyroid disorders. To investigate PCOS, laboratory tests should include total testosterone, DHEA-S (reserved for women with severe virilization), 17-Hydroxyprogesterone (to evaluate virilizing ovarian or adrenal tumors or CAH), prolactin, and thyroid-stimulating hormone (TSH).

MCQ's

1. Which hormone is primarily responsible for male libido
   - a) Estrogen b) Progesterone
   - c) Testosterone d) Oxytocin
2. What condition is characterized by a prolonged and painful erection lasting more than 4 hours
   - a) Peyronie's disease b) Priapism
   - c) Phimosis d) Erectile dysfunction
3. What is Peyronie's disease
   - a) A condition of the testes
   - b) A curvature of the penis due to fibrous scar tissue
   - c) An infection of the prostate
   - d) A type of hernia
4. What is the term for the inability to ejaculate despite normal erection and sexual arousal
   - a) Anejaculation b) Retrograde ejaculation
   - c) Premature ejaculation d) Delayed ejaculation
5. What condition is characterized by pain during intercourse
   - a) Anorgasmia b) Dyspareunia
   - c) Vaginismus d) Hypoactive sexual desire disorder
6. Which of the following is a common symptom of hypoactive sexual desire disorder
   - a) Pain during intercourse b) Lack of sexual fantasies
   - c) Inability to achieve orgasm d) Vaginal dryness
7. What is anorgasmia
   - a) Pain during intercourse b) Inability to achieve orgasm
   - c) Lack of sexual desire d) Vaginal dryness

8. What is the term for involuntary muscle spasms of the vaginal muscles that interfere with intercourse
   - a) Anorgasmia b) Dyspareunia
   - c) Vaginismus d) Hypoactive sexual desire disorder
9. What condition is characterized by the absence of sperm in the ejaculate
   - a) Azoospermia b) Oligospermia
   - c) Aspermia d) Teratozoospermia
10. Which diagnostic test is commonly used to assess male fertility
    - a) Complete blood count (CBC) b) Urinalysis
    - c) Semen analysis d) Electrocardiogram
11. What is the typical pH range of normal semen
    - a) 5.0-6.0 b) 6.5-7.5
    - c) 7.2-8.0 d) 8.5-9.5
12. What is the normal sperm concentration
    - a) 10 million/mL b) 15 million/mL
    - c) 20 million/mL d) 25 million/mL
13. What is the most common cause of female infertility
    - a) Uterine fibroids b) Tubal blockage
    - c) Ovulatory disorders d) Cervical issues
14. What is the primary function of the Anti-Mullerian Hormone (AMH) test in evaluating female infertility
    - a) Assessing uterine health b) Evaluating ovarian reserve
    - c) Measuring estrogen levels d) Detecting fallopian tube blockages
15. What is the effect of hyperprolactinemia on female fertility
    - a) Enhances ovulation b) Causes luteinisation
    - c) Suppresses ovulation d) Promotes endometrial growth

Answer Keys

1	c	2	b	3	b	4	a	5	b	6	b	7	b	8	c
9	a	10	c	11	c	12	b	13	c	14	b	15	c		

◆

TOPIC 50

Unmāda and Apasmāra

UNMĀDA

Introduction

Unmāda is one of the major Mānasika Vyādhi characterized by Samudbhrama (confusion or agitation) of Buddhi (intellect), Manas (mind) and Smṛtī (memory). Ācārya Caraka described Unmāda after Śoṣa but it had been manifested after Kuṣṭha due to destruction of Dakṣaprajāpati yajña. Because of endogenous nature and importance, Śoṣa has been explained after Kuṣṭha. Ācārya Suśruta described Unmāda after Apasmāra pratiṣedha because of Manogatasāmyāt (similarity in afflicting the mind) and Tulyacikitsitatvā (similarity in the treatment) as well as he described that if Unmāda is Apravṛddha (not excessively aggravated) and Taruṇa (in its early stage), then it is called as Mada. Due to Cikitsā sāmānyāt (similarity in the treatment), Ācārya Vāgbhaṭa explained Unmāda after Bhūta pratiṣedha. Ācārya Mādhavakara described that the features of Caturthamada is similar to that of Unmāda. Unmatta, Cittavibhrama, Mativibhrānti and Unmanā are the synonyms of Unmāda.

Nirūkti and Paribhāṣa

The term Unmāda is derived from the root 'Ut + Mad' suffixed with 'Ghañ' which means much to be mad or extreme madness or foolishness i.e. insanity.

- समुद्भ्रमं बुद्धिमनःस्मृतीनामुन्मादमागन्तुनिजोत्थमाहुः। (Ca.Ci.9/8)

That which causes Samudbhrama (confusion or agitation) of Buddhi (intellect), Manas (mind) and Smṛtī (memory) is known as Unmāda and it is of two types namely Nija (endogenous) and Āgantuja (exogenous).

- मदयन्त्युद्ध(द्र)ता दोषा यस्मादुन्मार्गमाश्रिताः । मानसोऽयमतो व्याधिरुन्माद इति कीर्तितः ॥ (Su.U.62/3)
- उन्मादो नाम मनसो दोषैरुन्मार्गगैर्मदः॥ (A.H.U.6/1)

That which causes upward movement of aggravated doṣas located in the heart producing derangement of mind is known as Unmāda.

- उद्गता वृद्धाः, अथवा ऊर्ध्वं हृदयं गताः, एतेनोत्पूर्वेणैव दोषाणां वृद्धत्वं विमार्गगत्वं च दर्शितम्। (Ma.M.N.20/1)

Madhukośa while commenting on this, he had mentioned that Udgatā means Vṛddhā or Ūrdhvaṃ hṛdaya gatāḥ i.e. vitiation of doṣas in the heart; Unmārgamāgātāḥ means Vimārgamāgatā manovahādhamanīranuprāptāḥ i.e. the aggravated doṣas moves upwards to reach Manovahādhamanīs and 'Mada' means 'Madayanti' meaning Manovibhrama, i.e. derangement or confusion of mind.

Therefore, Unmāda means the doṣas located in the heart gets aggravated and moves upwards to reach Manas through Manovahādhamanīs and produces Manovibhrama i.e. derangement or confusion of mind.

- उन्मादं पुनर्मनोबुद्धिसञ्ज्ञाज्ञानस्मृतिभक्तिशीलचेष्टाचारविभ्रमं विद्यात्॥ (Ca.Ni.7/5)

Unmāda is defined as Vibhrama (perversion or confusion) of Mana (mind), Buddhi (intellect), Saṅjñā (consciousness), Jñāna (knowledge), Smṛti (memory), Bhakti (Iccha - desire), Śīla (temper or attitude), Ceṣṭa (activities) and Ācāra (conduct).

- अत्र मनोविभ्रमाच्चिन्त्यानर्थान्न चिन्तयते, अचिन्त्यांश्च चिन्तयते; उक्तं हि- "मनसश्च चिन्त्यमर्थः" इति। बुद्धिविभ्रमात्तु नित्यमनित्यमिति, प्रियं चाप्रियमिति पश्यति; वचनं हि- "विषमाभिनिवेशो यो नित्यानित्ये प्रियाप्रिये। ज्ञेयः स बुद्धिविभ्रंशः, समं बुद्धिर्हि पश्यति" इति। सञ्ज्ञां ज्ञानं, तद्विभ्रमादग्न्यादिदाहं न बुध्यते; किंवा सञ्ज्ञा नामोल्लेखेन ज्ञानम्। स्मृतिविभ्रमात्तु न स्मरति, अयथावद्वा स्मरति। भक्तिरिच्छा, तद्विभ्रमाच्च यत्रेच्छा पूर्वमासीत्तत्रानिच्छा भवति। शीलविभ्रमादक्रोधनः क्रोधनो भवति। चेष्टाविभ्रमादनुचितचेष्टो भवति। आचारः शास्त्रशिक्षाकृतो व्यवहारः; तद्विभ्रमादशौचाद्याचरति। (Ca.Ca.Ni.7/5)

Ācārya Cakrapāṇi while commenting on this, he opines the following

- **Mano Vibhrama:** Manasaśca cintyamarthaḥ (thinking is the object of mind); due to perversion of mind, Cintyānarthānna cintayte (the patient does not think about relevant topics) and Acintyāṃśca cintayate (thinking up of irrelevant topics).
- **Buddhi Vibhrama:** Samaṃ buddhirhi paśyati (intellect foresees rightly); due to perversion of intellect, Viṣamābhiniveśo yo nityānitye priyāpriye (the patient does wrong determination between eternal and ephemeral as well as useful and harmful things).
- **Jñāna Vibhrama:** Saṅjñāṃ jñānaṃ (consciousness is knowledge); due to perversion of knowledge, Agnyādidāhaṃ na budhyate kiṃvā saṅjñā nāmollekhena jñānam (the patient does not feel burning sensation caused by fire or even he fails to write the word Saṅjñā).

- **Smṛti Vibhrama:** Due to perversion of memory, Na smarati athavāvadvā smarati (he does not remember anything or remembers things incorrectly).
- **Bhakti Vibhrama:** Bhaktirichhā (Bhakti is desire); due to pervasion of desire, Yatrecchā pūrvamāsīttatrānicchā bhavati (person develops dislikes for things liked previously).
- **Śīla Vibhrama:** Śīla means character; due to pervasion of character, Akrodhanaḥ krodhano bhavati (the one free from anger gets enraged).
- **Ceṣṭa Vibhrama:** Ceṣṭa means activity; due to pervasion of activity, Anucitaceṣṭo bhavati (the patient indulges in improper activities).
- **Ācāra Vibhrama:** Ācāra means conduct; due to pervasion of conduct, Śāstraśikṣākṛto vyavahāraḥ aśaucādyācarati (the patient does against the rules preached in scriptures such as observes non – cleanliness etc.).

Sāmānya Nidāna and Saṃprāpti

- भीरूणामुपक्लिष्टसत्त्वानामुत्सन्नदोषाणां समलविकृतोपहितान्यनुचितान्याहारजातानि वैषम्ययुक्तेनोपयोगविधिनोपयुञ्जानानां तन्त्रप्रयोगमपि विषममाचरतामन्याश्च शरीरचेष्टा विषमाः समाचरतामत्युपक्षीणदेहानां व्याधिवेगसमुद्भ्रमितानामुपहतमनसां वा कामक्रोधलोभहर्षभयमोहायास शोकचिन्तोद्वेगादिभिर्भूयोऽभिघाताभ्याहतानां वा मनस्युपहते बुद्धौ च प्रचलितायामभ्युदीर्णा दोषाः प्रकुपिता हृदयमुपसृत्य मनोवहानि स्रोतांस्यावृत्य जनयन्त्युन्मादम्॥ (Ca.Ni.7/4)

In those who are Bhīrūṇām (timid or shy), Upakliṣṭasattvanām (have distressed mind with predominance of Rajas and Tamas), Utsannadoṣāṇāṃ (have been afflicted with aggravated doṣas), Samalavikṛtopahitānyanucitānyāhāra (indulging in impure or dirty and unwholesome food substances or touched by unclean hands of person suffering from Kuṣṭha, Vyaṅga etc.), Vaiṣamyayuktenopayogavidhinopayuñjānānāṃ (neglecting the rules of dietetics), Tantraprayogamapi viṣamamācaratāmanyāśca (performing Tāntrika practices with wrong procedures), Śarīra ceṣṭā viṣamāḥ (performing abnormal physical activities), Samācaratāmatyupakṣīṇadehanāṃ (have been exceedingly emaciated), Vyādhivega samudbhramitānāmupahatamanasāṃ (have confused mind due to intensity of other disease) or Kāmakrodhalobhaharṣabhayamohāyāsaśokacintodvegādibhirbhūyo (mind is repeatedly afflicted with passion, anger, greed, exhilaration, fear, delusion, exertion, grief, worry) or Abhighātābhyāhatānāṇ manasyupahate (excessive physical or mental assault), in such condition, the Buddhi (intellect) becomes Pracalitāyāma (extremely bewildered or confused) and the aggravated doṣas spread towards the Hṛdaya (heart) covering the Manovaha srotas and produces the disease Unmāda.

- विरुद्धदुष्टाशुचिभोजनानि प्रधर्षणं देवगुरुद्विजानाम्। उन्मादहेतुर्भयहर्षपूर्वो मनोऽभिघातो विषमाश्च चेष्टाः॥ तैरल्पसत्त्वस्य मलाः प्रदुष्टा वुद्धेर्निवासं हृदयं प्रदूष्य। स्रोतांस्यधिष्ठाय मनोवहानि प्रमोहयन्त्याशु नरस्य चेतः॥ (Ca.Ci.9/4-5)

If the Alpasattva (weak minded) person indulges in Viruddhaduṣṭāśuci bhojanāni (contrary, polluted and impure food substances), Pradharṣaṇaṃ devagurudvijānām

(disrespect gods, teachers and Brāhmaṇas), Manoabhighāta (mental trauma) and Viṣamā ceṣṭāḥ (irregular activities), then the doṣas gets aggravated and it vitiate the Hṛdaya (heart) which is the abode of Buddhi (intellect) and it reaches the Manovaha srotas and produces the disease Unmāda in which the person's mind becomes bewildered quickly.

Saṃprāpti Ghaṭaka of Unmāda

Doṣa	Tridoṣas, Rajas and Tamas
Dūṣya	Manas and Rasādi Sapta dhātus especially Rasa and Rakta
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Manovaha srotas
Srotoduṣṭi	Saṅga, Vimārga gamana and Atipravṛtti
Sañcāra sthāna	Manovaha srotas
Adhiṣṭhāna	Manas, Indriya and Buddhi
Udbhava sthāna	Āmapakvāśaya and Hṛdaya
Vyakta sthāna	Manas and Sarva Śarīra
Rogamārga	Madhyama

Pūrvarūpa of Unmāda

- तस्येमानि पूर्वरूपाणि; तद्यथा- शिरसः शून्यता, चक्षुषोराकुलता, स्वनः कर्णयोः, उच्छ्वासस्याधिक्यम्, आस्यसंस्रवणम्, अनन्नाभिलाषारोचकाविपाकाः, हृद्ग्रहः, ध्यानायाससम्मोहोद्वेगाश्चास्थाने, सततं लोमहर्षः, ज्वरश्चाभीक्ष्णम्, उन्मत्तचित्तत्वम्, उदर्दित्वम्, अर्दिताकृतिकरणं च व्याधेः, स्वप्ने चाभीक्ष्णं दर्शनं भ्रान्तचलितानवस्थितानां रूपाणामप्रशस्तानां च तिलपीडकचक्राधिरोहणं वातकुण्डलिकाभिश्चोन्मथनं निमज्जनं च कलुषाणामम्भसामावर्ते चक्षुषोश्चापसर्पणमिति (दोषनिमित्तानामुन्मादानां पूर्वरूपाणि भवन्ति)॥ (Ca.Ni.7/6)
- मोहोद्वेगौ स्वनः श्रोत्रे गात्राणामपकर्षणम् । अत्युत्साहोऽरुचिश्चान्ने स्वप्ने कलुषभोजनम् ।
  वायुनोन्मथनं चापि भ्रमश्चक्रगतस्य वा । यस्य स्यादचिरेणैव उन्मादं सोऽधिगच्छति ॥ (Su.U.62/6-7)

Śirasaḥśūnyatā (emptiness in the head), Cakṣuṣorākulatā (perplexity in the eyes), Svanaḥkarṇayoḥ or Svanaḥ kṣotre (noise in the ears or tinnitus), Gātrāṇāmapakarṣaṇam (emaciation of the body), Atyutsāho (excessive enthusiasm), Ucchvāsasyādhikyam (excessive hard breathing), Āsyasaṃsravaṇam (excessive salivation in the mouth), Anannābhilāṣa (aversion to intake food), Arocaka (anorexia), Avipākā (indigestion), Hṛdgraha (spasm in the cardiac region), Dhyāna (meditation), Āyāsa (fatigue), Sammoha (delusion), Udvegāścasthāne (anxiety in improper situation), Satataṃ lomaharṣa (constant horripilation), Jvaraścābhīkṣṇam (frequent attacks of fever), Unmattacittatvam (fickle mindedness), Udarditvam (pressing type of pain in the upper part of body), Arditākṛtikaraṇaṃ ca vyādhe (facial expression as if affected with facial paralysis), Svapne cābhīkṣṇaṃ darśanaṃ bhrāntacalitānavasthitānāṃ rūpāṇāmapraśastānāṃ (frequent appearance of dreams of wandering, moving and unstable inauspicious objects), Tilapīḍakacakrādhirohaṇaṃ (riding over the wheel of an oil press), Bhramaścakragatasya (giddiness as if due to rotating on a wheel), Vātakuṇḍalikābhiśconmathana or Vāyunonmathanaṃ (being churned by whirl winds), Nimajjanaṃ kaluṣāṇāmambhasām (sinking in whirlpool of turbid waters), Kaluṣabhojana (eating dirty food) and Āvartecakṣuṣoścāpasarpaṇamiti (diminishing eyesight or defective vision) are the premonitory symptoms of Unmāda caused by doṣas

Sāmānya Lakṣaṇās of Unmāda

- ⊙ धीविभ्रमः सत्त्वपरिप्लवश्च पर्याकुला दृष्टिरधीरता च। अबद्धवाक्त्वं हृदयं च शून्यं सामान्यमुन्मादगदस्य लिङ्गम्॥ स मूढचेता न सुखं न दुःखं नाचारधर्मौ कुत एव शान्तिम्। विन्दत्यपास्तस्मृतिबुद्धिसञ्ज्ञो भ्रमत्ययं चेत इतस्ततश्च॥ (Ca.Ci.9/6-7)

Dhīvibhrama (bewildered intellect), Sattvapariplava (unsteady mind), Paryākulā dṛṣṭi (turbid or agitated eyes), Adhīratā (unsteadiness), Abaddhavāktvaṃ (incoherent speech) and Hṛdayaṃ ca śūnyaṃ (feeling of emptiness in the heart) are the general features of Unmāda.

Thus the person with Mūḍhacetā (confounding mind), does not know Sukha (pleasure) or Duḥkha (pain), Nācāradharma (does not know code of conduct) and Śāntim (does not get peace of mind anywhere) and his mind wavers here and there due to loss of Smṛti (memory), Buddhi (intellect) and Saṃjñā (consciousness).

Unmāda Bheda

- ⊙ .....उन्मादमागन्तुनिजोत्थमाहुः। (Ca.Ci.9/8)

Ācārya Caraka classified Unmāda in to two types broadly i.e. Nija (endogenous) and Āgantuja (exogenous).

- ⊙ इह खलु पञ्चोन्मादा भवन्ति; तद्यथा- वातपित्तकफसन्निपातागन्तुनिमित्ताः॥ (Ca.Ni.7/3)

Ācārya Caraka classified Unmāda in to five types namely Vātaja, Pittaja, Kaphaja, Sannipātja and Āgantuja Unmāda.

- ⊙ एकैकशः समस्तैश्च दोषैरत्यर्थमूर्च्छितैः । मानसेना च दुःखेन स पञ्चविध उच्यते ॥ विषाद्भवति षष्ठश्च यथास्वं तत्र भेषजम् । स चाप्रवृद्धस्तरुणो मदसञ्ज्ञां बिभर्ति च ॥ (Su.U.62/4-5)
- ⊙ उन्मादाः षट् पृथग्दोषनिचयाधिविषोद्भवाः। (A.H.U.6/1)

Ācārya Suśruta and Vāgbhaṭa classified Unmāda in to six types namely Vātaja, Pittaja, Kaphaja, Sannipātja, Duḥkhaja (Ādhija) and Viṣaja Unmāda

Viśeṣa Unmāda Lakṣaṇās

Vātaja Unmāda

- ⊙ रूक्षाल्पशीतान्नविरेकधातुक्षयोपवासैरनिलोऽतिवृद्धः। चिन्तादिजुष्टं हृदयं प्रदूष्य बुद्धिं स्मृतिं चाप्युपहन्ति शीघ्रम्॥ अस्थानहासस्मितनृत्यगीतवागङ्गविक्षेपणरोदनानि। पारुष्यकार्श्यारुणवर्णताश्च जीर्णे बलं चानिलजस्य रूपम्॥ (Ca.Ci.9/9-10)

Due to Rūkṣālpaśītānna (intake of food which is non-unctuous, little in quantity and cold in property), Vireka (excessive evacuation), Dhātukṣaya (depletion of body tissues) and Upavāsa (fasting), the Vāta gets excessively aggravated and it affects the Hṛdaya (heart) which is already afflicted with Cintādi (worries etc.) and quickly impede the Buddhi (intellect) and Smṛti (memory).

Hāsa (laughing), Smita (smiling), Nṛtya (dancing), Gīta (singing), Vāk (speaking), Aṅgavikṣepaṇa (tossing the body parts) and Rodana (weeping) at improper place, Pāruṣya (roughness), Kārśya (emaciation), Aruṇavarṇatā (reddish discoloration of the skin) and Jīrṇe balaṃ (these features become strong after digestion of food) are the clinical features of Vātaja Unmāda.

- ⊙ रूक्षच्छविः परुषवाग्धमनीततो वा शीतातुरः कृशतनुः स्फुरिताङ्गसन्धिः ।
  आस्फोटयत्यटति गायति नृत्यशीलो विक्रोशति भ्रमति चाप्यनिलप्रकोपात् ॥ (Su.U.62/8)

Rukṣacchavi (dry skin), Paruṣavāk (harsh voice), Dhamanītato (prominence of veins) Śītāturaḥ (feeling of cold), Kṛśatanuḥ (emaciated and thin body), Sphuritāngasandhiḥ (twitching or throbbing of joints) along with Āsphoṭayatyaṭati (moves), Gāyati (sings), Nṛtyaśīlo (dances), Vikroṣati (cry out) and Bhramati (wanders about) are the features of Vātaja Unmāda.

Pittaja Unmāda

- ⊙ अजीर्णकट्वम्लविदाह्यशीतैर्भोज्यैश्चितं पित्तमुदीर्णवेगम्। उन्मादमत्युग्रमनात्मकस्य हृदि श्रितं पूर्ववदाशु कुर्यात्॥ अमर्षसंरम्भविनग्नभावाः सन्तर्जनातिद्रवणौष्ण्यरोषाः । प्रच्छायशीतान्नजलाभिलाषाः पीता च भाः पित्तकृतस्य लिङ्गम्॥ (Ca.Ci.9/11-12)

Ajīrṇa (indigestion) and Kaṭvamlavidāhyaśītairbhojyaiścita (intake of food which is pungent, sour, food substances causing burning sensation and hot in potency) leads to aggravation of accumulated Pitta and it afflicts the Hṛdaya (heart) who is Anātmakasya (devoid of self-control) and manifests serious type of Unmāda instantaneously with the pathogenesis described earlier.

Amarṣa (intolerance), Saṃrambha (arrogance), Vinagnabhāvāḥ (nakedness), Santarjana (threatening), Atidravaṇauṣṇya (excessive heat in the body), Roṣāḥ (anger), Pracchāyaśītānnajalābhilāṣāḥ (desire for dense shade, cold food and water) and Pitā (yellowish complexion) are the clinical features of Pittaja Unmāda.

- ◉ तृट्स्वेददाहबहुलो बहुभुग्विनिद्रश्छायाहिमानिलजलान्तविहारसेवी ।
  तीक्ष्णो हिमाम्बुनिचयेऽपि स वह्निशङ्की पित्ताद्दिवा नभसि पश्यति तारकाश्च ॥ (Su.U.62/9)

Bahulatṛṭ (excessive thirst), Bahulasveda (excessive perspiration), Bahuladāha (excessive burning sensation), Bahubhuk (voracious eating), Vinidra (sleeplessness), Chāyāhimānilajalāntavihārasevī (likes shade, cool breeze and walking or wandering in the watery places), Tikṣṇo (Kopanaḥ - anger), Himāmbunicayeapi sa vahniśaṅkī (suspects fire even inside ice-water) and Divā nabhasi paśyati tārakā (sees stars in sky during daytimes) are the features of Pittaja Unmāda.

Kaphaja Unmāda

- ◉ सम्पूरणैर्मन्दविचेष्टितस्य सोष्मा कफो मर्मणि सम्प्रवृद्धः। बुद्धिं स्मृतिं चाप्युपहत्य चित्तं प्रमोहयन् सञ्जनयेद्विकारम्॥
  वाक्चेष्टितं मन्दमरोचकश्च नारीविविक्तप्रियताऽतिनिद्रा। छर्दिश्च लाला च बलं च भुङ्क्ते नखादिशौक्ल्यं च कफात्मकस्य॥
  (Ca.Ci.9/13-14)

Due to Saṃpūraṅa (excessive nourishment) and Mandaviceṣṭitasya (slow activities), the Kapha along with Ūṣmā (heat) afflicts the Marma (vital organs) and impairs the Buddhi (intellect) and Smṛti (memory) and thereby infatuate the Citta (mind) and manifests the disease Unmāda.

Vākceṣṭitaṃ mandam (slow speech and activities), Arocaka (anorexia), Nārīviviktapriyatā (liking for women and loneliness), Atinidrā (excessive sleep), Chardi (vomiting), Lālāsrāva (salivation), Bhuṅkte ca balaṃ (these features become strong after intake of food) and Nakhādiśauklyaṃ (whiteness of nails etc.) are the clinical features of Kaphaja Unmāda.

- ◉ छर्द्यग्निसादसदनारुचिकासयुक्तो योषिद्विविक्तरतिरल्पमतिप्रचारः ।
  निद्रापरोऽल्पकथनोऽल्पभुगुष्णसेवी रात्रौ भृशं भवति चापि कफप्रकोपात् ॥ (Su.U.62/10)

Chardi (vomiting), Agnisāda (weak digestive fire), Sadana (malaise), Aruci (anorexia), Kāsa (cough), Yoṣidviviktarati (likes women and loneliness), Alpamatipracāraḥ (less mental and physical activities), Nidra (sleeps excessively), Aparoalpakathano (speaks less), Alpabhuk (eats very little quantity), Uṣṇasevī (uses hot things) and Rātrau bhṛśaṃ (these features aggravates at the night time) are the features of Kaphaja Unmāda.

Sannipātaja Unmāda

- ◉ यः सन्निपातप्रभवोऽतिघोरः सर्वैः समस्तैः स च हेतुभिः स्यात्। सर्वाणि रूपाणि बिभर्ति तादृग्विरुद्धभैषज्यविधिर्विवर्ज्यः॥ (Ca.Ci.9/15)

In Sannipātaja Unmāda, all the three doṣas gets aggravated simultaneously due to their respective causative factors described above and it is considered as Atighora (severe and serious disease) and produces all the features of the three doṣas. Due to contradictory treatment principles, this condition should be discarded.

- ◉ सर्वात्मके पवनपित्तकफा यथास्वं संहर्षिता इव च लिङ्गमुदीरयन्ति ॥ (Su.U.62/11)

In case of Sannipātaja Unmāda, all the three doṣas i.e. Vāta, Pitta and Kapha are aggravated and exhibit their respective features.

Mānasika duḥkhaja Unmāda

- ◉ चौरैनरिन्द्रपुरुषैरभिस्तथाऽन्यैर्वित्रासितस्य धनबान्धवसङ्क्षयाद्वा ।
  गाढं क्षते मनसि च प्रियया रिरंसोर्जायेत चोत्कटतरो मनसो विकारः ॥
  चित्रं स जल्पति मनोनुगतं विसञ्ज्ञो गायत्यथो हसति रोदिति मूढसञ्ज्ञः । (Su.U.62/12-13)
- ◉ धनकान्तादिनाशेन दुःसहेनाभिषङ्गवान्। पाण्डुर्दीनो मुहुर्मुह्यन् हाहेति परिदेवते॥
  रोदित्यकस्मान्म्रियते तद्गुणान् बहु मन्यते। शोकक्लिष्टमना ध्यायन् जागरूको विचेष्टते॥ (A.H.U.6/15-16)

The person whose mind is Gāḍhaṃkṣate (excessively injured) due to Caurairnarendrapuruṣairabhistathāanyair vitrāsitasya (frightened by thieves, king's men, enemies and others), Dhanabāndhavasaṃkṣayādvā (loss of wealth and of relatives), Priyayārimso (bereavement by the disappointed love) produces Utkaṭataro manaso vikāra (severe mental diseases). In this condition, the patient Citraṃ sa jalpati manonugataṃ visaṃjño (speaks inarticulately on many subjects unconsciously) and also Gāyatyatho hasati rodati mūḍhasaṃjñaḥ (sings, laughs or weeps with confounding mind).

Viṣaja Unmāda

- ◉ रक्तेक्षणो हतबलेन्द्रियभाः सुदीनः श्यावाननो विषकृतेऽथ भवेत् परासुः ॥ (Su.U.62/13)
- ◉ विषेण श्याववदनो नष्टच्छायाबलेन्द्रियः। वेगान्तरेऽपि सम्भ्रान्तो रक्ताक्षस्तं विवर्जयेत्॥ (A.H.U.6/17)

Raktekṣaṇā (redness of the eyes), Hatabalendriyabhāḥ (loss of strength, senses and complexion), Sudīnaḥ (extreme malaise), Śyāvānano (dark brownish discoloration of face) are the features of Viṣaja Unmāda and even it causes Parāsuḥ (death).

Āgantuja Unmāda

Nidāna

- ◉ देवर्षिगन्धर्वपिशाचयक्षरक्षःपितृणामभिधर्षणानि। आगन्तुहेतुर्नियमव्रतादि मिथ्याकृतं कर्म च पूर्वदेहे॥ (Ca.Ci.9/16)

Abhigharṣaṇā (possession) by the Devas (gods), Ṛṣis (sages), Gandharvas (celestial musician), Piśācas (demons), Yakṣa (spirit or ghost or spiritual apparition), Rakṣaḥ (evil demons) and Pitṛṇā (forefather's spirits); Āgantuheturniyamavratādi mithya kṛta (unmethodical performance of religious duties, vows etc.) and Pūrvadeha karma (deeds of past life) are the causes of the Āgantuja Unmāda.

Mode of entry of Graha

- ⦿ अदूषयन्तः पुरुषस्य देहं देवादयः स्वैस्तु गुणप्रभावैः। विशन्त्यदृश्यास्तरसा यथैव च्छायातपौ दर्पणसूर्यकान्तौ॥ (Ca.Ci.9/18)

Devādyaḥ svaistu guṇaprabhāvaiḥ (own qualities of god etc.,) swiftly enter into the Puruṣadeha (body of the person) with Adūṣayanta (very less damaging or without damaging) and it remains Adṛśya (invisibly) like that of Chāyātapau darpaṇasūryakāntau (image enters the mirror and sunrays enters in sun-stone).

- ⦿ तत्रायमुन्मादकराणां भूतानामुन्मादयिष्यतामारम्भविशेषो भवति; तद्यथा- अवलोकयन्तो देवा जनयन्त्युन्मादं, गुरुवृद्धसिद्धमहर्षयोऽभिशपन्तः, पितरो दर्शयन्तः, स्पृशन्तो गन्धर्वाः, समाविशन्तो यक्षाः, राक्षसास्त्वात्मगन्धमाघ्रापयन्तः, पिशाचाः पुनरारुह्य वाहयन्तः॥ (Ca.Ni.7/12)

Avalokayanto devā janayantyūnmadāṃ (god produce Unmāda by their looks), Guruvṛddhasiddhamaharṣayoabhiśapantaḥ (teachers, elders, accomplished persons and great sages by curses), Pitaro darśayantaḥ (ancestors by their appearance), Spṛśanto gandharvā (Gandharvas by their touch), Samāviśanto yakṣā (Yakṣa by entering in the body or mind of the person), Rākṣasāstvātmagandhamāghrāpayantaḥ (Rākṣasas by making the person to smell their body odour) and Piśācāḥ punarāruhya vāhayantaḥ (Piśācas by riding and driving the persons).

Pūrvarūpa of Āgantuja Unmāda

- ⦿ तत्र देवादिप्रकोपनिमित्तेनागन्तुकोन्मादेन पुरस्कृतस्येमानि पूर्वरूपाणि भवन्ति; तद्यथा- देवगोब्राह्मणतपस्विनां हिंसारुचित्वं, कोपनत्वं, नृशंसाभिप्रायता, अरतिः, ओजोवर्णच्छायाबलवपुषामुपतप्तिः, स्वप्ने च देवादिभिरभिभर्त्सनं प्रवर्तनं चेति; ततोऽनन्तरमुन्मादाभिनिर्वृत्तिः॥ (Ca.Ni.7/11)

Devagobrāhmaṇatapasvināṃ hiṃsārucitvaṃ (desire to inflict gods, cows, Brāhmaṇas and ascetics), Kopanatvaṃ (anger), Nṛśaṃsābhiprāyatā (cruel intention), Arati (distress), Ojovarṇacchāyābalavapuṣāmupatapti (impairment of Ojas, complexion, lustre, strength and body) and Svapne ca devādibhirabhibhartsanaṃ pravartanaṃ (intimidating and incitement by the gods etc., in dreams) are the premonitory features of the Āgantuja Unmāda.

Sāmānya Lakṣaṇās of Āgantuja Unmāda

- ⦿ अमर्त्यवाग्विक्रमवीर्यचेष्टो ज्ञानादिविज्ञानबलादिभिर्यः। उन्मादकालोऽनियतश्च यस्य भूतोत्थमुन्मादमुदाहरेत्तम्॥ (Ca.Ci.9/17)

Vāk (speech), Vikrama (valor), Vīrya (potentiality) and Ceṣṭa (activity) are similar to Amartya (immortals), Jñānādivijñānabalādibhiryaḥ (having similar knowledge, understanding, strength etc., of immortals) and Unmādakāloaniyata (irregular time of occurrence of disease Unmāda) are the example of features of Bhūtotthamunmāda (Unmāda caused by evil spirits).

- ⦿ अत्यात्मबलवीर्यपौरुषपराक्रमग्रहणधारणस्मरणज्ञानवचनविज्ञानानि, अनियतश्चोन्मादकालः॥ (Ca.Ni.7/13)

Atyātmabala (superhuman strength), Vīrya (potency), Pauruṣa (manliness), Parākrama (courage), Grahaṇa (grasping), Dhāraṇa (retention), Smaraṇa (memory), Jñāna (knowledge), Vacana (speech) and Vijñānāni (critical understanding) and Aniyataśconmāda kāla (uncertainty of time of paroxysm of Unmāda).

Viśeṣa Lakṣaṇās of Āgantuja Unmāda

Unmāda caused by Deva

- ⦿ सौम्यदृष्टिं गम्भीरमधृष्यमकोपनमस्वप्नभोजनाभिलाषिणमल्पस्वेदमूत्रपुरीषवातं शुभगन्धं फुल्लपद्मवदनमिति देवोन्मत्तं विद्यात्; (Ca.Ci.9/20)

Saumyadṛṣṭiṃ (gentle look), Gambhīram (serious or intense), Adhṛṣyam (invisible) Kopanamasvapnabhojanābhilāṣiṇa (free from anger, sleep and desire for food), Alpasvedamūtrapuriṣavāta (having diminished sweat, urine, faeces and flatus), Śubhagandhaṇ (emitting good aroma) and Phullapadmavadana (having face like blossomed lotus) are the features of Unmāda caused by Deva.

Unmāda caused by Guru

- ⦿ गुरुवृद्धसिद्धर्षीणामभिशापाभिचाराभिध्यानानुरूपचेष्टाहारव्याहारं तैरुन्मत्तं विद्यात्; (Ca.Ci.9/20)

Ceṣṭāhāravyāhāra (having activities, diet and speech) corresponding to Abhiśāpa (curse), Abhicāra (magical spell) and Abhidhyāna (meditation) of Guruvṛddhasiddharṣī (teachers, elders, accomplished person and sages) are the features of Unmāda caused by Guru..

Unmāda caused by Pitṛ

- ⦿ अप्रसन्नदृष्टिमपश्यन्तं निद्रालुं प्रतिहतवाचमनन्नाभिलाषमरोचकाविपाकपरीतं च पितृभिरुन्मत्तं विद्यात्; (Ca.Ci.9/20)

Aprasannadṛṣṭi (unhappy look), Apaśyantaṃ (not seeing anywhere), Nidrāluṃ (excessive sleep) Pratihatavācam (interrupted speech), Anannābhilāsa (absence of desire for food), Arocaka (anorexia) and Avipāka (indigestion) are the features of Unmāda caused by Pitṛ.

Unmāda caused by Gandharva

- ⦿ (चण्डं साहसिकं तीक्ष्णं गम्भीरमधृष्यं) मुखवाद्यनृत्यगीतान्नपानस्नानमाल्यधूपगन्धरतिं रक्तवस्त्रबलिकर्महास्यकथानुयोगप्रियं शुभगन्धं च गन्धर्वोन्मत्तं विद्यात्; (Ca.Ci.9/20)

Caṇḍa (violent or cruel), Sāhasika (ferocious), Tīkṣṇa (sharpness), Gambhīram (serious or intense), Adṛṣyam (invisible), who is liking for Mukhavādya (musical instruments played by mouth), Nṛtya (dancing), Gīta (singing), Annapāna (diet and drinks), Snāna (bath) Mālya (garlands), Dhūpa (incense), Gandharatiṃ (perfumes), Raktavastra (red garments), Balikarma (sacrificial oblations), Hāsya (laughing), Kathānuyoga (humorous talks) and Śubhagandha (emits pleasant aroma from the body) are the features of Unmāda caused by Gandharva.

Unmāda caused by Yakṣa

- ⊙ असकृत्स्वप्नरोदनहास्यं नृत्यगीतवाद्यपाठकथान्नपानस्नानमाल्यधूपगन्धरतिं रक्तविप्लुताक्षं द्विजातिवैद्यपरिवादिनं रहस्यभाषिणं च यक्षोन्मत्तं विद्यात्; (Ca.Ci.9/20)

Asakṛtsvapnarodanahāsyaṃ (sleeps, cries and laughs frequently or repeatedly), liking of Nṛtya (dancing), Gīta (singing), Vādya (playing musical instruments), Pāṭha (reciting scriptures), Katha (telling stories), Annapāna (diet and drinks), Snāna (bath) Mālya (garlands), Dhūpa (incense), Gandharatiṃ (perfumes), Raktaviplutākṣa (red and tearful eyes), Dvijātivaidyaparivādinaṃ (abusing or blaming Brāhmaṇas and physicians) and Rahasyabhāṣīṇaṃ (disclosing the secrets) are the features of Unmāda caused by Yakṣa.

Unmāda caused by Rākṣasa

- ⊙ नष्टनिद्रमन्नपानद्वेषिणमनाहारमप्यतिबलिनं शस्त्रशोणितमांसरक्तमाल्याभिलाषिणं सन्तर्जकं च राक्षसोन्मत्तं विद्यात्; (Ca.Ci.9/20)

Naṣṭanidra (sleeplessness), Annapānadveṣi (aversion to diet and drinks), Anāhāramapyatibalinaṃ (very strong even without taking food), Śāstraśoṇitamāṃsaraktamālyābhilāṣiṇaṃ (liking for weapons, blood, flesh and red garlands), and Santarjakaṃ (ferociousness) are the features of Unmāda caused by Rākṣasa

Unmāda caused by Brahmarākṣasa

- ⊙ प्रहासनृत्यप्रधानं देवविप्रवैद्यद्वेषावज्ञाभिः स्तुतिवेदमन्त्रशास्त्रोदाहरणैः काष्ठादिभिरात्मपीडनेन च ब्रह्मराक्षसोन्मत्तं विद्यात्; (Ca.Ci.9/20)

Prahāsanṛtyapradhāna (engaged in laughing and dancing), Devavipravaidyadveṣāvajñābhiḥ (hatred and disobedience to gods, Brāhmaṇas and physicians), Stutivedamantraśāstrodāharaṇai (reading the praises, hymns and other scriptures), Kāṣṭhādibhirātmapīḍanena (injuring himself with piece of wood etc.) are the features of Unmāda caused by Brahmarākṣasa.

Unmāda caused by Piśāca

- ⊙ अस्वस्थचित्तं स्थानमलभमानं नृत्यगीतहासिनं बद्धाबद्धप्रलापिनं सङ्करकूटमलिनरथ्याचेलतृणाश्मकाष्ठाधिरोहणरतिं भिन्नरूक्षस्वरं नग्नं विधावन्तं नैकत्र तिष्ठन्तं दुःखान्यावेदयन्तं नष्टस्मृतिं च पिशाचोन्मत्तं विद्यात्॥ (Ca.Ci.9/20)

Asvasthacittaṃ (fickle mindedness), Sthānamalabhamānaṃ (does not find peace to live), Nṛtyagītahāsinaṃ (dances, sings and laughs frequently), Baddhābaddhapralāpinaṃ (delirious with coherent or incoherent speech), Saṅkarakūṭamalinarathyācelatṛṇāśma kāṣṭhādhirohaṇaratiṃ (liking for climbing over heap of garbage, mountain, dirty paths, cloth, grass, stone and wood), Bhinnarūkṣasvara (hoarse and rough voice), Nagnaṃ (remains naked), Vidhāvantaṃ (running here and there), Naikatra tiṣṭhantaṃ (not standing in one place), Duḥkhānyāvedayantaṃ (always narrates his worries and sorrows) and Naṣṭasmṛtiṃ (loss of memory) are the features of Unmāda caused by Piśāca.

Sādhyāsādhyata of Unmāda

- ⊙ ते तु खलु निजागन्तुविशेषेण साध्यासाध्यविशेषेण च प्रविभज्यमानाः पञ्च सन्तो द्वावेव भवतः। तौ च परस्परमनुबध्नीतः कदाचिद्यथोक्तहेतुसंसर्गात्। तयोः संसृष्टमेव पूर्वरूपं भवति, संसृष्टमेव च लिङ्गम्। तत्रासाध्यसंयोगं साध्यासाध्यसंयोगं चासाध्यं विद्यात्, साध्यं तु साध्यसंयोगम्। तस्य साधनं साधनसंयोगमेव विद्यादिति॥ (Ca.Ni.7/18)

Even though it is mentioned that the Unmāda is of five types, it is actually only of two types Nija (endogenous) and Āgantuja (exogenous) or Sādhya (curable) and Asādhya (incurable). Sometimes, due to combination of Nidāna (causative factors), they manifest in a combined form. Such form is exhibited in their Pūrvarūpa (premonitory symptoms) and Liṅga (clinical features). In such cases, combination of Asādhya (incurable ones) and also combination of Sādhya (curable) with Asādhya (incurable one) becomes Asādhya (incurable); whereas the combination of Sādhya (curable ones) becomes Sādhya (curable), which should be treated with combination of therapeutic measures.

- ⊙ सर्वेष्वपि तु खल्वेषु यो हस्तावुद्यम्य रोषसंरम्भान्निःशङ्कमन्येष्वात्मनि वा निपातयेत् स ह्यसाध्यो ज्ञेयः; तथा यः साश्रुनेत्रो मेढ्रप्रवृत्तरक्तः क्षतजिह्वः प्रस्रुतनासिकश्छिद्यमानचर्माऽप्रतिहन्यमानवाणिः सततं विकूजन् दुर्वर्णस्तृषार्तः पूतिगन्धश्च स हिंसार्थिनोन्मत्तो ज्ञेयः; तं परिवर्जयेत्॥ (Ca.Ci.9/22)

In aforesaid types of Āgantuja Unmāda, if the person Hastāvudyamya roṣa saṃrambhānniḥśaṅka manyeṣvātmani vānipātayet sa hyasādhyo jñeyaḥ (raises his hand with anger, arrogance and without any hesitation beats others or himself should be considered as incurable). Similarly, the one who has Sāśrunetra (tearful eyes), Meḍhrapravṛttaraktaḥ (bleeding from penis), Kṣatajihvaḥ (injured tongue), Prasrutanāsika (running nose), Cidyamānacarmā (bruised skin), Apratihanyamānavāṇiḥ (continuously speaking), Satataṃ vikūjan (constant groaning), Durvarṇa (deranged complexion), Tṛṣārta (excessive thirst) and Pūtigandha (putrid smell emitting from their body) should be considered as Unmāda seized with demon desirous for Hiṃsa (killing) and it should be discarded from the treatment.

In Contemporary science

Unmāda cannot be correlated to a particular disease in Contemporary science. The psychiatric or neuropsychiatric diseases like mood or bipolar disorders, schizophrenia, major depressive disorders, etc., come under the umbrella of Unmāda. Research scholars correlated Doṣaja and Āgantuja Unmāda as follows

- ⊙ **Vātaja Unmāda** with schizophrenia, hebephrenia, catatonia, dementia, bipolar disorder with psychotic features, organic or secondary psychoses.
- ⊙ **Pittaja Unmāda** with manic psychosis, manic episode due to general medical condition, bipolar disorder co-morbid with hyperthyroidism or Graves' disease
- ⊙ **Kaphaja Unmāda** with major depressive disorder, myxedema / hypothyroidism with depression.

- **Āgantuja Unmāda** with paranoid schizophrenia

APASMĀRA

Introduction

Apasmāra is one among the Aṣṭamahā gada and it is characterized by Samplava (destruction or derangement) of Smṛtī (memory), Buddhi (intellect) and Sattva (mind). Ācārya Caraka described Apasmāra after Unmāda because after destruction of Dakṣaprajāpati yajña, Apasmāra manifested after Unmāda by Vividhabhūtāśucisaṃsparśāt (contact with various impure or unclean objects and creatures). And also he described that Apasmāra is of four types except Āgantuja (exogenous), because exogenous causes do not manifest Apasmāra independently like Unmāda.

Ācārya Suśruta described Apasmāra after Amānuṣopasarga pratiṣedha because of Manaḥpradusti sāmānyāt (similarity in afflicting the mind). Due to Cikitsāsāmānyāt (similarity in the treatment), Ācārya Vāgbhaṭa explained Apasmāra after Unmāda pratiṣedha. Smṛtidvaṃsa, Aṅgavikṛti, Lālādha, Lolāṅga and Bhūtavikriyā are the synonyms of Apasmāra.

Nirūkti and Paribhāṣa

The word 'Apasmāra' is derived from the root 'Apa + Smṛ' suffixed with 'Ṇic' which means loss of memory.

- अपस्मारयति स्मरणं विलोपयति । (Vācaspatyam)

That which causes Vilopayati (destruction) of Smaraṇa (memory) is known as Apasmāra.

- अपस्मर्य्यते पूर्व्ववृत्तं विस्मर्य्यतेऽनेन । (SKD)

That which causes Pūrvavṛttaṃ vismaryate (forgotting the before chosen) is known as Apasmāra.

- स्मृतेरपायो अपस्मार उच्यते। (Aru.A.H.U.7/1-5)

That which causes Smṛterapāya (diminution or loss of memory) is known as Apasmāra.

- अपगतः स्मारो यस्मिन् रोगे सोऽपस्मारः । (Ḍa.Su.U.61/1-2)

The disease which causes Apagata (gone off) of Smaraṇa (memory) is known as Apasmāra.

- स्मृतिर्भूतार्थविज्ञानमपश्च परिवर्जने । अपस्मार इति प्रोक्तस्ततोऽयं व्याधिरन्तकृत् ॥ (Su.U.61/3)

The word Smṛti means Bhūtārthavijñāna (the knowledge of anything that has really happened or really exists) and Apa means absence or loss. Hence, this fatal disease is termed as Apasmāra in which the individual loses the reminiscence.

- स्मृतेरपगमं प्राहुरपस्मारं भिषग्विदः। तमःप्रवेशं बीभत्सचेष्टं धीसत्त्वसम्प्लवात्॥ (Ca.Ci.10/3)

The physicians define Apasmāra as Apagamana i.e. disappearance of Smṛti (memory) associated with Tamaḥpraveśa (feeling of entering into darkness), Bībhatsaceṣṭa (disgusting activities) and Dhīsattvasaṃplavāt (derangement of the intellect and the mind).

Sāmānya Nidāna and Saṃprāpti

- त एवंविधानां प्राणभृतां क्षिप्रमभिनिर्वर्तन्ते; तद्यथा- रजस्तमोभ्यामुपहतचेतसामुद्भ्रान्त विषमबहुदोषाणां समलविकृतोपहितान्यशुचीन्यभ्यवहारजातानि वैषम्ययुक्तेनोपयोग विधिनोपयुञ्जानानां तन्त्रप्रयोगमपि च विषममाचरतामन्याश्च शरीरचेष्टा विषमाः समाचरतामत्युपक्षयाद्वा दोषाः प्रकुपिता रजस्तमोभ्यामुपहतचेतसामन्तरात्मनः श्रेष्ठतममायतनं हृदयमुपसृत्योपरि तिष्ठन्ते, तथेन्द्रियायतनानि च। तत्र चावस्थिताः सन्तो यदा हृदयमिन्द्रियायतनानि चेरिताः कामक्रोधभयलोभमोहहर्षशोकचिन्तोद्वेगादिभिः सहसाऽभिपूरयन्ति, तदा जन्तुरपस्मरति॥ (Ca.Ni.8/4)

Apasmāra manifests quickly in persons who has Rajastamobhyāmupahatacetasām (inflictions of mind by Rajas and Tamas), Udbhrāntaviṣamabahudoṣāṇāṃ (doṣas raised irregularly and excessively aggravated), Samalavikṛtopahitānyaśucīnyabhyavahārajātāni (consuming dirty, contaminated, damaged and unclean food substances), Vaiṣamyayuktenopayogavidhinopayuñjānānāṃ (following the improper dietetic rules), Tantraprayogamapi ca viṣamamācaratāmanyāśca (practicing improper techniques), Śarīraceṣṭā viṣamāḥ (practicing improper postures and movements of the body) and Samācaratāmatyupakṣayādva (associated with excessive wasting or emaciation), the doṣas gets aggravated and afflicts the person having mind already affected by Rajas and Tamas. The aggravated doṣas spreading towards the Hṛdaya which is the Śreṣṭhamamāyatana (excellent abode) of Antarātmanaḥ (inner self) as well as Indriyas (sense organs) and stay there in latent stage. While staying there, the doṣas gets aggravated further by Kāma (passion), Krodha (anger), Bhaya (fear), Lobha (greed), Moha (delusion), Harṣa (exhilaration), Śoka (grief), Cinta (worries), Udvegādi (agitation etc.) and suddenly fill up the Hṛdaya (heart) and Indriyāyatana (abode of sense organs) and produce the disease Apasmāra.

- विभ्रान्तबहुदोषाणामहिताशुचिभोजनात् । रजस्तमोभ्यां विहते सत्त्वे दोषावृते हृदि॥
  चिन्ताकामभयक्रोधशोकोद्वेगादिभिस्तथा। मनस्यभिहते नृणामपस्मारः प्रवर्तते॥
  धमनीभिः श्रिता दोषा हृदयं पीडयन्ति हि। सम्पीड्यमानो व्यथते मूढो भ्रान्तेन चेतसा॥
  पश्यत्यसन्ति रूपाणि पतति प्रस्फुरत्यपि। जिह्वाक्षिभ्रूः स्रवल्लालो हस्तौ पादौ च विक्षिपन्॥
  दोषवेगे च विगते सुप्तवत् प्रतिबुद्ध्यते। (Ca.Ci.10/4-8)

Due to intake of Ahitāśucibhojanāt (unwholesome and unclean food substances), Rajastamobhyāṃ vihate sattve (in whom the Sattva (attributes of mind) is subdued by Rajas and Tamas), Doṣāvṛte hṛdi (heart is enclosed with aggravated doṣas) and Cintākāmabhayakrodhaśokodvegādi manasyabhihate (mind is afflicted by worry, passion, fear, anger, grief, agitation, etc.), the accumulated doṣas gets excessively aggravated and spreads upwards in the dhamanīs (vessels) and afflicts the Hṛdaya and causes disturbances in its functions and manifests the disease Apasmāra.

In which the person suffers from Bhrāntena cetasā (confusion of mind), Paśyatyasanti rūpāṇī (visualizes non-existent forms), Patati (falls down), Prasphuratyapi jihvākṣibhrūḥ (tremulous tongue, eyes and eyebrows), Sravallālo (excessive salivation), Hastau pādau ca vikṣipan (contracted hands and legs) and Doṣavege ca vigate suptavat pratibuddhyate (once the attacks are over, he regains consciousness as if awakening from sleep).

- ⦿ मिथ्यातियोगेन्द्रियार्थकर्मणामभिसेवनात् । विरुद्धमलिनाहारविहारकुपितैर्मलैः ॥
  वेगनिग्रहशीलानामहिताशुचिभोजिनाम् । रजस्तमोभिभूतानां गच्छतां च रजस्वलाम् ॥
  तथा कामभयोद्वेगक्रोधशोकादिभिर्भृशम् । चेतस्यभिहते पुंसामपस्मारोऽभिजायते ॥ (Su.U.61/4-6)

Due to Mithyātiyogendriyārthakarmaṇāmabhisevanāt (improper and excessive use of sense objects and actions), Viruddhamalināhāravihāra (intake of incompatible and unhygienic food substances and activities),Veganigraha (suppression of natural urges), Śīlānāmahitāśucibhojanāt (habitual intake of unwholesome and unclean food substances), Rajastamobhibhūtānāṃ (mind subdued by Rajas and Tamas), Gacchatāṃ ca rajasvalām (indulging in coitus with menstruating woman) and often afflicted with Kāmabhayodvegakrodhaśokādi (passion, fear, agitation, anger, grief, etc.), the doṣas gets aggravated and it afflicts the Cetas (mind) and gives rise to Apasmāra.

- ⦿ सञ्ज्ञावहेषु स्रोतःसु दोषव्याप्तेषु मानवः । रजस्तमःपरीतेषु मूढो भ्रान्तेन चेतसा ॥
  विक्षिपन् हस्तपादं च विजिह्मभ्रूर्विलोचनः । दन्तान् खादन् वमन् फेनं विवृताक्षः पतेत् क्षितौ ॥
  अल्पकालान्तरं चापि पुनः सञ्ज्ञां लभेत सः । सोऽपस्मार इति प्रोक्तः स च दृष्टश्चतुर्विधः ॥ (Su.U.61/8-10)

When the Saṃjñāvaha srotas become pervaded by doṣas in an individual under the influence of Rajas and Tamas, he suffers from Bhrāntena cetasā (confusion of mind), Vikṣipan Hastapādaṃ (contracted hands and legs), Vijihmabhrūrvilocana (crooked eyebrows and eye sight), Dantān khādan (bites teeth), Vaman phenaṃ (emits froth from the mouth), Vivṛtākṣaḥ patet (falls down with wide open eyes) and Alpakālāntaraṃ cāpi punaḥ saṃjñāṃ labhate (he regains consciousness after a short period).

Saṃprāpti Ghaṭaka of Apasmāra

Doṣa	Tridoṣas, Rajas and Tamas
Dūṣya	Rasa
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhya janya Āma
Srotas	Saṃjñāvaha srotas
Srotoduṣṭi	Saṅga and Atipravṛtti
Sañcāra sthāna	Hṛdaya and Dhamanīs
Adhiṣṭhāna	Hṛdaya and Indriyāyatana
Udbhava sthāna	Āmapakvāśaya and Hṛdaya
Vyakta sthāna	Sarva Śarīra
Rogamārga	Madhyama

Pūrvarūpa

- ⦿ तस्येमानि पूर्वरूपाणि भवन्ति; तद्यथा- भ्रूव्युदासः सततमक्ष्णोर्वैकृतमशब्दश्रवणं लालासिङ्घाणप्रस्रवणमनन्नाभिलाषणमरोचकाविपाकौ हृदयग्रहः कुक्षेराटोपो दौर्बल्यमस्थिभेदोऽङ्गमर्दो मोहस्तमसो दर्शनं मूर्च्छा भ्रमश्चाभीक्ष्णं स्वप्ने च मदनर्तनव्यधनव्यथनवेपनपतनादीनीति॥ (Ca.Ni.8/6)

Bhrūvyudāsaḥ (destruction or displacement of eyebrows), Satatamakṣṇorvaikṛtam (constant abnormal movement of eyes), Aśabdaśravaṇam (hearing of non-existent sounds), Lālāsinghāṇa prasravaṇa (excessive salivation and nasal discharge), Anannābhilāṣaṇam (loss of desire for food), Arocaka (anorexia), Avipāka (indigestion), Hṛdayagraha (stiffness of the cardiac region), Kukṣerāṭopa (distension of abdomen with gurgling sound), Daurbalya (weakness), Asthibheda (cracking pain in the bones), Aṅgamarda (body pain), Moha (delusion), Tamaso darśanaṃ (feeling of darkness before eyes), Mūrcchā (fainting), Bhrama (giddiness) and Abhīkṣṇaṃ svapne ca madanartanavyadhanavyathanavepanapatanādī (frequent dreams of intoxication, dancing, piercing, aching, trembling and falling) are the premonitory features of Apasmāra.

- ⦿ हृत्कम्पः शून्यता स्वेदो ध्यानं मूर्च्छा प्रमूढता । निद्रानाशश्च तस्मिंस्तु भविष्यति भवन्त्यथ ॥ (Su.U.61/7)

Hṛtkampaḥ śūnyatā (tremors and feeling of emptiness in the cardiac region), Sveda (perspiration), Dhyāna (dullness), Mūrcchā (fainting), Pramūḍhatā (inertness bewildered) and Nidrānāśa (loss of sleep) are the premonitory features of Apasmāra.

Apasmāra Bheda

- ⦿ इह खलु चत्वारोऽपस्मारा भवन्ति वातपित्तकफसन्निपातनिमित्ताः॥ (Ca.Ni.8/3)
- ⦿ पृथग्दोषैः समस्तैश्च वक्ष्यते स चतुर्विधः॥ (Ca.Ci.10/8)
- ⦿ वातपित्तकफैर्नृणां चतुर्थः सन्निपाततः । (Su.U.61/11)

Apasmāra is classified in to four types namely, Vātaja, Pittaja, Kaphaja and Sannipātaja Apasmāra.

Sāmānya Lakṣaṇās of Apasmāra

- ⦿ अपस्मारं पुनः स्मृतिबुद्धिसत्त्वसम्प्लवाद्बीभत्सचेष्टमावस्थिकं तमः प्रवेशमाचक्षते॥ (Ca.Ni.8/5)

Apasmāra is characterized by Āvasthikaṃ tamaḥpraveśa (transient loss of consciousness) associated with Bibhatsaceṣṭa (loathsome activities such as froth coming out of mouth and twisting body parts) due to Smṛtibuddhisattvasamplavāt (derangement of memory, intellect and mind).

- हृदि तोदस्तृडुत्क्लेदस्त्रिष्वप्येतेषु सङ्ख्यया । प्रलापः कूजनं क्लेशः प्रत्येकं तु भवेदिह ॥ (Su.U.61/16)

Hṛditoda (pricking pain in the cardiac region), Tṛḍ (thirst) and Utkleda (nausea) are the specific symptoms of Vātaja, Pittaja and Kaphaja Apasmāra respectively; Pralāpa (delirium), Kūjana (cooing) and Kleśa (distress) are found in all types of Apasmāra.

Viśeṣa Lakṣaṇās

Vātaja Apasmāra

- अभीक्ष्णमपस्मरन्तं, क्षणेन सञ्ज्ञां प्रतिलभमानम्, उत्पिण्डिताक्षम्, असाम्ना विलपन्तम्, उद्वमन्तं फेनम्, अतीवाध्मातग्रीवम्, आविद्धशिरस्कं, विषमविनताङ्गुलिम्, अनवस्थितपाणिपादम्, अरुणपरुषश्यावनखनयनवदनत्वचम्, अनवस्थितचपलपरुषरूक्षरूपदर्शिनं, वातलानुपशयं, विपरीतोपशयं च वातेनापस्मरन्तं विद्यात् ॥ (Ca.Ni.8/8(1))

Abhīkṣṇamapsmarantaṃ (frequent episodes of unconsciousness), Kṣaṇena saṃjñām pratilabhamānam (regaining consciousness quickly), Utpiṇḍitākṣam (bulging eyes), Asāmnā vilapantam (incoherent speech), Udvamantaṃ phenam (vomiting of froth), Atīvādhmātagrīvam (rigid or fixed neck), Āviddhaśiraskaṃ (twisted or oblique position of head), Viṣamavinatāṅgulim (irregularly contracted fingers), Anavasthitapāṇipādam (unstable arms and legs), Aruṇaparuṣaśyāvanakhanayanavadanatvacam (reddishness, dryness and dark brownish discoloration of nails, eyes, face and skin), Anavasthitacalaparuṣarūkṣarūpadarśinam (vision of unstable, fickle, coarse and dry objects), Vātalānupaśayaṃ (unsuitability to Vāta aggravating factors) and Viparītopaśayaṃ (suitability to opposite i.e. Vāta pacifying factors) are the features of Vātaja Apasmāra.

- कम्पते प्रदशेद्दन्तान् फेनोद्वामी श्वसित्यपि। परुषारुणकृष्णानि पश्येद्रूपाणि चानिलात्। (Ca.Ci.10/9)

Vātaja Apasmāra is characterized by Kampate (trembles), Pradaśeddantān (biting teeth), Phenodvāmī (emits froth from the mouth), Śvasityapi (sighing) and Paruṣāruṇakṛṣṇāni paśyedrupāṇi (visualizes the forms as rough, reddish and black in color).

- वेपमानो दशन् दन्तान् श्वसन् फेनं वमन्नपि ॥ यो ब्रूयाद्विकृतं सत्त्वं कृष्णं मामनुधावति ।
  ततो मे चित्तनाशः स्यात् सोऽपस्मारोऽनिलात्मकः ॥ (Su.U.61/11-12)

The features of Vātaja Apasmāra are Vepamāno (trembles), Daśan dantān (biting teeth), Śvasan (sighing), Phenaṃ vamannapi (emits froth from the mouth), Brūyādvikṛtaṃ sattvaṃ kṛṣṇaṃ māmanudhāvati (describes that some strange black creature is chasing him) and then he loses his consciousness (Cittanāśa).

Pittaja Apasmāra

- अभीक्ष्णमपस्मरन्तं क्षणेन सञ्ज्ञां प्रतिलभमानम्, अवकूजन्तम्, आस्फालयन्तं भूमिं, हरितहारिद्रताम्रनखनयनवदनत्वचं, रुधिरोक्षितोग्रभैरवादीप्तरुषितरूपदर्शिनं, पित्तलानुपशयं, विपरीतोपशयं च पित्तेनापस्मरन्तं विद्यात् ॥ (Ca.Ni.8/8(2))

Abhīkṣṇamapsmarantaṃ (frequent episodes of unconsciousness), Kṣaṇena saṃjñāṃ pratilabhamānam (regaining consciousness quickly), Avakūjantam (cooing sound), Āsphālayantaṃ bhūmiṃ (striking the ground), Haritahāridratāmranakhanayanavadanatvacam (greenish, yellowish and coppery discoloration of nails, eyes, face and skin), Rudhirokṣitograbhairavādīptaruṣitarūpadarśinaṃ (vision of bloody, agitated, dreadful, burning and angry objects), Pittalānupaśayaṃ (unsuitability to Pitta aggravating factors) and Viparītopaśayaṃ (suitability to opposite i.e. Pitta pacifying factors) are the features of Pittaja Apasmāra.

- पीतफेनाङ्गवक्त्राक्षः पीतासृग्रूपदर्शनः। सतृष्णोष्णानलव्याप्तलोकदर्शी च पैत्तिकः॥ (Ca.Ci.10/10)

Pittaja Apasmāra is characterized by Pītephena (emits yellow froth from the mouth), Pīte Aṅgavaktrākṣa (yellowish discoloration of the body parts, face and eyes), Pītāsṛgrūpadarśana (visualizes the forms as yellow and red) and it is associated with Tṛṣṇā (thirst), Uṣṇa (heat sensation) and Analavyāptalokadarśī (visualizes whole world as seized with fire).

- तृट्तापस्वेदमूर्च्छार्तो धुन्वन्नङ्गानि विह्वलः । यो ब्रूयाद्विकृतं सत्त्वं पीतं मामनुधावति ॥
  ततो मे चित्तनाशः स्यात् स पित्तभव उच्यते । (Su.U.61/13-14)

The features of Pittaja Apasmāra are Tṛṭ (thirst), Tāpa (heat), Sveda (perspiration), Mūrcchā (fainting), Dhunvannaṅgāni (shaking the body parts), Vihvalaḥ (distress), Brūyādvikṛtaṃ sattvaṃ pītaṃ māmanudhāvati (describes that some strange yellow creature is chasing him) and then he loses his consciousness (Cittanāśa).

Kaphaja Apasmāra

- चिरादपस्मरन्तं, चिराच्च सञ्ज्ञां प्रतिलभमानं, पतन्तम्, अनतिविकृतचेष्टं, लालामुद्वमन्तं, शुक्लनखनयनवदनत्वचं, शुक्लगुरुस्निग्धरूपदर्शिनं, श्लेष्मलानुपशयं, विपरीतोपशयं च श्लेष्मणाऽपस्मरन्तं विद्यात्॥ (Ca.Ni.8/8(3))

Ciradapsmarantaṃ (delayed episodes of unconsciousness), Cirācca saṃjñāṃ pratilabhamānam (delayed regaining of consciousness), Patantam (falling down), Anativikṛtaceṣṭaṃ (not very distorted activities), Lālāmudvamantaṃ (vomiting of saliva), Śuklanakhanayanavadanatvacam (whitish discoloration of nails, eyes, face and skin), Śuklagurusnigdharūpadarśinaṃ (vision of white, heavy and unctuous objects), Śleṣmalānupaśayaṃ (unsuitability to Śleṣma aggravating factors) and Viparītopaśayaṃ (suitability to opposite i.e. Śleṣma pacifying factors) are the features of Śleṣmaja Apasmāra.

- शुक्लफेनाङ्गवक्त्राक्षः शीतो हृष्टाङ्गजो गुरुः। पश्यञ्छुक्लानि रूपाणि श्लैष्मिको मुच्यते चिरात्॥ (Ca.Ci.10/11)

Śleṣmaja Apasmāra is characterized by Śuklaphena (emits white froth from the mouth), Śukla Aṅgavaktrākṣa (whitish discoloration of the body parts, face and eyes), Śīta (cold sensation), Hṛṣṭa (exhilaration), Aṅgajo guruḥ (heaviness of the body parts) and Paśyañcuklāni rūpāṇi (visualizes the forms as white) and Mucyate cirāt (recovers after a long period).

- ⦿ शीतहृल्लासनिद्रार्तः पतन् भूमौ वमन् कफम् ॥ यो ब्रूयाद्विकृतं सत्त्वं शुक्लं मामनुधावति ।
  ततो मे चित्तनाशः स्यात् सोऽपस्मारः कफात्मकः ॥ (Su.U.61/14-15)

The features of Kaphaja Apasmāra are Śita (feeling cold), Hṛllāsa (nausea), Nidrārta (sleep), Patan bhūmau (falls on ground), Vaman kapham (vomiting mucus), Brūyādvikṛtaṃ sattvaṃ śuklaṃ māmanudhāvati (describes that some strange white creature is chasing him) and then he loses his consciousness (Cittanāśa).

Sannipātaja Apasmāra

- ⦿ समवेतसर्वलिङ्गमपस्मारं सान्निपातिकं विद्यात्, तमसाध्यमाचक्षते ॥ (Ca.Ni.8/8(4))
- ⦿ सर्वलिङ्गसमवायः सर्वदोषप्रकोपजे । (Su.U.61/17)

Samaveta sarvaliṅga or Sarvaliṅga samavāya (combined features of all the three doṣas) are manifested in case of Sannipātaja Apasmāra and it is Asādhya (incurable).

- ⦿ सर्वैरेतैः समस्तैस्तु लिङ्गैर्ज्ञेयस्त्रिदोषजः। अपस्मारः स चासाध्यो यः क्षीणस्यानवश्च यः॥ (Ca.Ci.10/12)

In Sannipātaja Apasmāra, the features of all the three doṣas are manifested in combined form and it is Asādhya (incurable). And Apasmāra that occurs in Kṣīṇa (emaciated person) and Anava (in chronic stage) is also said to be Asādhya (incurable).

Apasmāra Prakopakāla

- ⦿ पक्षाद्वा द्वादशाहाद्वा मासाद्वा कुपिता मलाः। अपस्माराय कुर्वन्ति वेगं किञ्चिदथान्तरम्॥ (Ca.Ci.10/13)

The aggravated doṣas produce paroxysm of Apasmāra once in fifteen days, twelve days or a month, however, this may occur one after the other with very short period.

Āgantuja Apasmāra

- ⦿ तेषामागन्तुरनुबन्धो भवत्येव कदाचित्, तमुत्तरकालमुपदेक्ष्यामः।
  तस्य विशेषविज्ञानं यथोक्तलिङ्गैर्लिङ्गाधिक्यमदोषलिङ्गानुरूपं च किञ्चित्॥ (Ca.Ni.8/9)
- ⦿ यस्यानुबन्धस्त्वागन्तुर्दोषलिङ्गाधिकाकृतिः। दृश्येत तस्य कार्यं स्यादागन्तून्मादभेषजम्॥ (Ca.Ci.10/53)

Āgantuja Apasmāra does not manifest independently, hence Ācāryās have not explained it as a separate type. But, at times Āgantuja (exogenous) factors are associated with these (aforesaid four) conditions and their specific features are the additional symptoms which have not been manifested due to vitiated doṣas. In such condition, the measures prescribed for Āgantuja Unmāda should be adopted.

Sādhyāsādhyata of Apasmāra

- ⦿ अपस्मारो हि वातेन पित्तेन च कफेन च। चतुर्थः सन्निपातेन प्रत्याख्येयस्तथाविधः॥
  साध्यांस्तु भिषजः प्राज्ञाः साधयन्ति समाहिताः। तीक्ष्णैः संशोधनैश्चैव यथास्वं शमनैरपि॥
  यदा दोषनिमित्तस्य भवत्यागन्तुरन्वयः। तदा साधारणं कर्म प्रवदन्ति भिषग्विदः॥ (Ca.Ni.8/12-14)

Apasmāra is caused by vitiation of Vāta, Pitta, Kapha and Sannipata. And the fourth one (Sannipataja) is Asādhya (incurable). The physician should carefully treat the curable ones with Tīkṣṇa saṃśodhana (strong purificatory therapies) and respective Śamana (pacificatory) therapies. When the Apasmāra caused by doṣas gets associated with Āgantuja (exogenous) factors, then the physician should prescribe the Sādhāraṇa karma (general line of treatment) mentioned for Doṣaja Apasmāra (i.e. Vamanādi) and Āgantuja Apasmāra (i.e. Balīmaṅgalādi).

In Contemporary science

Apasmāra can be predominantly characterised by conditions like epilepsy in Contemporary science (Please refer Topic 53)

MCQ's

1. Unmāda is due to

a) Smṛti Vibhrama
b) Icchā Vibhrama
c) Ācāra Vibhrama
d) All of the above

2. Arditākṛtikaraṇaṃ ca vyādhe is the Pūrvarūpa of

a) Unmāda
b) Apasmāra
c) Mūrcchā
d) All of the above

3. Hṛdayaṃ ca śūnyaṃ is the feature of

a) Unmāda
b) Apasmāra
c) Both A and B
d) All of the above

4. Aggravation of the symptoms of Kaphaja Unmāda takes place

a) Bhuṅkte ca balaṃ
b) Jīrṇe balaṃ
c) Pācanakāle balaṃ
d) None of the above

5. Redness of the eyes is seen in

a) Duḥkahaja Unmāda
b) Viṣaja Unmāda
c) Ādhija Unmāda
d) All of the above

6. According to Vāgbhaṭa, Unmāda is of .... types

a) 3	b) 4
c) 5	d) 6

7. Smṛtidvaṃsa is the synonym of

a) Unmāda	b) Apasmāra
c) Viṣāda	d) Both A and B

8. In Apasmāra, Prakupita doṣās spreads towards

a) Manas	b) Buddhi
c) Hṛdaya	d) Śākha

9. Apasmāra is of .... types

a) 3	b) 4
c) 5	d) 6

10. Tamaḥpraveśa is the feature of

a) Unmāda	b) Apasmāra
c) Both A and B	d) All of the above

11. Kṣaṇena saṃjñāṃ pratilabhamānam is the feature of

a) Vātaja Apasmāra	b) Pittaja Apasmāra
c) Kaphaja Apasmāra	d) Both A and B

Answer Keys

1	d	2	a	3	a	4	a	5	b	6	d
7	b	8	c	9	b	10	b	11	d		

◆

TOPIC 51

Viṣāda

Introduction

Viṣāda is also one among the Manovikāra and it is one of the characteristics of Tāmasā Prakṛti. Ācārya Caraka described Viṣāda is one among the Nānātmajā Vātavyādhi and he considered as Rogavardhanānāṃ śreṣṭam (the prime factor for aggravators of diseases). Ācārya Cakrapāṇi described that Śarīramanaso glānikara (the factors which causes lassitude or fatigue or exhaustion of body and mind) is Viṣāda. Ācārya Suśruta described Viṣāda in the Sūtra sthāna while describing the four types of diseases i.e. Āgantava (traumatic), Śārīra (somatic), Mānasā (psychic) and Svabhāva (natural). Ācārya Ḍalhaṇa mentioned that Viṣāda is one of the types of Dveṣa. Avasāda, Dainya and Kheda are used as synonyms of Viṣāda.

Nirūkti and Paribhāṣa of Viṣāda

The term 'Viṣāda' is derived from the root 'Viṣad' which means depression of spirits or insensibility or disappointment.

- विषादो मनोदैन्यम् । (Ga.Ca.Sū.20/11)

That which causes Manodainya (depression or miserable state of mind) is called Viṣāda.

- विषादिताग्लानिः। (He.A.H.Ni.2/17)

That which causes Glāniḥ (fatigue or lassitude) is called Viṣāda.

- असिद्धिभयाद्विविधेषु कर्मसु सादोऽप्रवृत्तिः विषादः । (Ḍa.Su.Sū.1/25(3))
- विषादोऽनुष्ठेये आत्मनोऽशक्तताज्ञानम् ॥ (Ca.Su.Sū.1/25(3))

That which causes Asiddhibhayādvividheṣu karmasu sādoapravṛttiḥ (loss of initiation in different activities due to fear or failure) or Anuṣteye ātmanośaktatājñānam (incapable to engaging in self activities) is called Viṣāda.

- ⦿ वाक्कायचित्तावसादो विषादः। (Ḍa.Su.Ka.3/18-22)

That which causes Vākkāyacittāvasāda (lassitude or fatigue of speech, body and mind) is called Viṣāda.

- ⦿ विषादिता-विषण्णत्वं दुःख़ितत्वम्। (Aru.A.H.Ni.2/17)

That which causes Viṣaṇṇatva (sadness or languor) and Duḥkhitatvam (grieved or unhappiness) is called Viṣāda.

- ⦿ अविषादकरं शरीरमनसोरग्लानिकरम्। (Ca.Ca.Vi.8/94)
- ⦿ अवसादः चित्तदेहयोर्ग्लानिः। (Ḍa.Su.Sū.15/13)

That which causes Śarīramanasoraglānikara (the factors which does not cause lassitude or fatigue or exhaustion of body and mind) is known as Aviṣāda. Therefore, it can be taken as the factor which causes Śarīramanaso glāni or Cittadehayorglāni (lassitude or fatigue or exhaustion of body and mind) is known as Viṣāda or Avasāda.

Nidāna and Saṃprāpti of Viṣāda

- ⦿ तत्त्रिविधम्- असात्म्येन्द्रियार्थसंयोगः, प्रज्ञापराधः, परिणामश्चेति॥ (Ca.Ni.1/3)

There is no specific causative factor of Viṣāda mentioned in the classics. As it is originated from the aggravation of Rajas and Tamas, general causative factor of Manāsika rogas such as Asātmyendriyārtha saṃyoga (unwholesome conjunction of sense organs with their objects), Prajñāparādha (intellectual error) and Pariṇāma (consequences) can be taken as causative factors of Viṣāda.

- ⦿ वीभत्सनं मनस उद्वेगकारकं । (Ca.Ca.Sū.11/37)

Ācārya Cakrapāṇi described that Bībhatsanaṃ (loathsome or disgusting factors) causes Mānasa udvega (depression or anxiety of mind).

- ⦿ मात्रयाऽप्यभ्यवहृतं पथ्यं चान्नं न जीर्यति। चिन्ताशोकभयक्रोधदुःखशय्याप्रजागरैः॥ (Ca.Vi.2/9)

Even the most wholesome food taken in proper quantity does not get properly digested due to Cintā (worry), Śoka (grief), Bhaya (fear), Krodha (anger), Duḥkhaśayyā (uncomfortable bed) and Prajāgaraī (vigil).

- ⦿ यातयामं गतरसं पूति पर्युषितं च यत् । उच्छिष्टमपि चामेध्यं भोजनं तामसप्रियम् ॥ (B.G.2/17/10)

As it is mentioned that Viṣāda is one of the characteristics of Tāmasā Prakṛti, hence the factors which increases the Tamo guṇa may be taken as causative factors of Viṣāda i.e. Yātayāmāṃ (spoiled), Gatarasaṃ (tasteless), Pūti (putrid), Paryuṣita (decomposed), Ucchiṣṭa (stale) and Āmedhya (foul and impure) food substances.

By these factors, the Vāta gets aggravated especially Prāna, Udāna and Vyāna which are responsible for Utsāha (enthusiasm), Harṣa (exhilaration); Smṛti (memory) and Ceṣṭa (movement) respectively along with Sādhaka pitta which is responsible for proper functions of Buddhimedhābhimāna (intellect, wisdom and arrogance) and Tarpaka Kapha which is responsible for Indriyatarpaṇa karma (functions of satiating the sense organs) and spreads towards the Hṛdaya (heart) covering the Manovaha srotasas and produces the disease Viṣāda.

Saṃprāpti Ghaṭaka of Viṣāda

Doṣa	Vāta Pradhāna Tridoṣa; Rajas and Tamas
Dūṣya	Manas and Rasādi dhātus
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Manovaha and Rasavaha Srotas
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Manovaha srotas
Adhiṣṭhāna	Manas, Hṛdaya
Udbhava sthāna	Āmapakvāśaya and Hṛdaya
Vyakta sthāna	Manas and Sarva Śarīra
Rogamārga	Madhyama

Viṣāda Lakṣaṇās

The clinical features of Viṣāda are Manodainya (depression or miserable state of mind), Glāniḥ (fatigue or lassitude), Asiddhibhayādvividheṣu karmasu sādoapravṛttiḥ (loss of initiation in different activities due to fear or failure), Anuṣṭeye ātmanośaktatājñānam (incapable to engaging in self activities), Vākkāyacittāvasāda (lassitude or fatigue of speech, body and mind), Viṣaṇṇatva (sadness or languor), Duḥkhitatvam (grieved or unhappiness) and Śarīramanaso glāni or Cittadehayorglāni (lassitude or fatigue or exhaustion of body and mind).

And also the features described by Arjuna are mentioned in Viṣāda yoga chapter of Bhagavadgīta may be taken as features of Viṣāda, i.e.

- ⦿ सीदन्ति मम गात्राणि मुखं च परिशुष्यति । वेपथुश्च शरीरे मे रोमहर्षश्च जायते ।
  गाण्डीवं स्रंसते हस्तात्त्वक्चैव परिदह्यते ॥ न च शक्नोभ्यवस्थातुं भ्रमतीव च मे मनः ॥ (B.G.1/28-30)

Sīdanti mama gātraṇi (weakening of limbs), Mukhaṃ ca pariśuṣyati (dryness of the mouth), Vepathuśca śarire (trembling of the whole body), Romaharṣa (horripilation), Gāṇḍīvaṃ sraṃsate hastāt (the bow Gāṇḍīva is slipping from the hand), Tvakcaiva paridahyate (skin is burning), Na ca śaknobyavasthātuṃ (unable to keep composed) and Bhramatīva ca me manaḥ (unsteady mind).

In Contemporary science

Viṣāda can be predominantly characterised by conditions like depression respectively in contemporary science. (Please refer Topic 53).

MCQ's

1. According to Ḍalhaṇa, Viṣāda is a type of

   a) Dveṣa b) Krodha
   c) Dainya d) Kheda

2. Viṣāda causes

   a) Manodainya b) Viṣaṇṇatva
   c) Glāniḥ d) All of the above

3. Which Doṣa is mainly associated with Viṣāda

   a) Vāta b) Pitta
   c) Kapha d) Sannipātaja

4. Which synonym is NOT associated with Viṣāda?

   a) Avasāda b) Dainya
   c) Kheda d) Prasāda

5. Which disease causes Vākkāyacittāvasāda

   a) Unmāda b) Apasmāra
   c) Viṣāda d) All of the above

Answer Keys

1	a	2	d	3	a	4	d	5	c

◆

TOPIC 52

Mūrcchā and Sannyāsa

Introduction

Mūrcchā (fainting) and Sannyāsa (unconsciousness) are the psychosomatic diseases afflicting the Saṃjñavaha srotas along with Rasavaha and Raktavaha srotas. The severity of the diseases in terms of Hetu (etiology), Liṅga (symptoms) and Upaśānti (pacification) extends in the consecutive order.

Nidāna and Saṃprāpti of Mūrcchā and Sannyāsa

◉ यदा तु रक्तवाहीनि रससञ्ज्ञावहानि च। पृथक् पृथक् समस्ता वा स्रोतांसि कुपिता मलाः॥
मलिनाहारशीलस्य रजोमोहावृतात्मनः। प्रतिहत्यावतिष्ठन्ते जायन्ते व्याधयस्तदा॥
मदमूर्च्छायसन्न्यासास्तेषांविद्याद्विचक्षणः।यथोत्तरंबलाधिक्यंहेतुलिङ्गोपशान्तिषु॥(Ca.Sū.24/25-27)

The person who indulges in Malināhāra śīla (habitual intake of impure food) and whose Ātman (self) is covered with Rajas and Moha (Tamas), the vitiated Doṣas, individually or all together obstruct the Rakta, Rasa and Saṃjñavaha srotas, gets located there and produces Mada, Mūrcchā and Sannyāsa. The severity of the diseases in terms of Hetu (etiology), Liṅga (symptoms) and Upaśānti (pacification) extends in the consecutive order.

◉ दुर्बलं चेतसः स्थानं यदा वायुः प्रपद्यते। मनो विक्षोभयञ्जन्तोः सञ्ज्ञां सम्मोहयेत्तदा॥
पित्तमेवं कफश्चैवं मनो विक्षोभयन्नृणाम्। सञ्ज्ञां नयत्याकुलतां विशेषश्चात्र वक्ष्यते॥ (Ca.Sū.24/28-29)

When the aggravated Vāta, reaches the Durbala cetasaḥ sthānaṃ (weak part of mind), it causes Mano vikṣobaya (agitation of the mind), then produces Saṃjñaṃ saṃmoha (unconsciousness). Similarly the aggravated Pitta and Kapha also agitate the mind and produces unconsciousness.

Saṃprāpti Ghaṭaka of Mūrcchā and Sannyāsa

Doṣa	Pitta Pradhāna Tridoṣa
Dūṣya	Rasa, Rakta and Tamas
Agni	Jaṭharāgni Dhātvāgni māndhya
Āma	Agnimāndhya janya Āma
Srotas	Saṃjña, Rasa and Raktavaha
Srotoduṣṭi	Saṅga
Adhiṣṭhāna	Manas
Udbhava Sthāna	Āmāśaya
Sañcāra Sthāna	Saṃjña, Rasa Raktavaha
Vyakta sthāna	Saṃjñavaha Nāḍi
Rogamārga	Bāhya and Abhyantara

MŪRCCHĀ

- ⊙ मूर्च्छयतीति सञ्ज्ञानाशं करोति । (Ca.Ca.Sū.26/43)

That which causes loss of consciousness is called Mūrcchā.

- ⊙ मूर्च्छा पित्ततमःप्राये ॥ (B.P.M.18/19)

Mūrcchā is predominantly caused by Pitta doṣa and Tamas doṣa

- ⊙ मूर्च्छा मोहसंज्ञा विख्याता, यतः मोहो मूर्च्छेति तामाहुः इत्यादि ॥ (Āḍ.Śā.P.7/31)

Mūrcchā is concerned with Moha (unconsciousness)

- ⊙ नष्ट सुखदुःखज्ञाने नरः काष्ठवत्पतति तां मोहो मूर्च्छेति प्राहुरित्यन्वयः ॥ (B.P.M.18/4)

Mūrcchā is a condition in which the person loses the knowledge of Sukha (happiness) and uḥkha (unhappiness); and Kāṣṭhvatpatati tāṃ moho (falls unconsciously like wooden log).

Paryāya (synonyms)

- ⊙ संज्ञोपघातो मूर्च्छायो मूर्च्छा स्यान्मूर्च्छनं तथा । कश्मलं प्रलयो मोहः सन्यासस्तु मृतोपम ॥ (B.P.M.18/3-4)

Saṃjñopaghāta, Mūrcchāya, Mūrcchanaṃ, Kaśmalaṃ, Pralaya and Moha are the synonyms of Mūrcchā.

Nidāna and Saṃprāpti of Mūrcchā

- ⊙ क्षीणस्य वहुदोषस्य विरुद्धाहारसेविनः । वेगाघातादभीघाताद्धीनसत्त्वस्य वा पुनः ॥
  करणायतनेषूग्रा वाह्येष्वाभ्यन्तरेषु च । निविशन्ते यदा दोषास्तदा मूर्च्छन्ति मानवाः ॥ (Su.U.46/3-4)

Persons who are Kṣīṇa (emaciated) with Hīna satva (mentally weak) and Bahu doṣa (doṣas are abundant), when indulge in Viruddha āhāra (incompatible diets), Vegādhāraṇa (suppression of natural urges) and Abhighāta (injury), the doṣas get aggravated and located in both Bāhya (external) and Ābhyantara (internal) seats of mind and produces the disease Mūrcchā.

- ⊙ रजोमोहाहिताहारपरस्य स्युस्त्रयो गदाः॥ रसासृक्ऽऽचेतनावाहिस्रोतोरोधसमुद्भवाः।
  मदमूर्च्छायसन्न्यासा यथोत्तरबलोत्तराः॥ (A.H.Ni.6/24-25)

Consuming food influenced by Rajas and Mohā may lead to the manifestation of three conditions, namely Mada, Mūrcchā, and Sannyāsa. These conditions result in the obstruction of the Rasa, Rakta, and Cetana Vaha srotas. It is noteworthy that these conditions tend to progress in severity, with each succeeding condition being more potent than its predecessor.

- ⊙ सञ्ज्ञावहासु नाडीषु पिहितास्वनिलादिभिः । तमोऽभ्युपैति सहसा सुखदुःखव्यपोहकृत् ॥
  सुखदुःखव्यपोहाच्च नरः पतति काष्ठवत् । मोहो मूर्च्छेति तां प्राहुः षड्विधा सा प्रकीर्तिता ॥ (Su.U.46/6-7)

The aggravated Vātādi doṣas obstructs the Saṃjñavaha Nāḍī, leading sudden increase in the Tamo guṇa which destroys the Sukhaduḥkhavyapohakṛt (perception of happiness and unhappiness). The person who had lost his knowledge of Sukha (happiness) and Duḥkha (unhappiness) falls unconsciously like Kāṣṭhvatpatati (wooden log). This condition is called Moha or Mūrcchā and it is of 6 types

Pūrvarūpa (premonitory symptoms)

- ⊙ हृत्पीडा जृम्भणं ग्लानिः सञ्ज्ञानाशो बलक्षयः । सर्वासां पूर्वरूपाणि, यथास्वं तां विभावयेत् ॥ (B.P.M.18/7)
- ⊙ हृत्पीडा जृम्भणं ग्लानिः सञ्ज्ञानाशो बलस्य च । सर्वासां पूर्वरूपाणि, यथास्वं ता विभावयेत् ॥ (Su.U.46/5)

Hṛtpīḍā (pain in the cardiac region), Jṛmbhaṇaṃ (yawning), Glāni (fatigue), Saṃjña Nāśa (loss of sensory perception) and Bala Kṣaya (loss of strength) are the premonitory symptoms of Mūrcchā.

Bheda and Lakṣaṇās

- ⊙ मूर्च्छा चतुर्विधा ज्ञेया वातपित्तकफैः पृथक् । चतुर्थी सन्निपातेन ॥ (Śā.P.7/31)
- ⊙ वातादिभिः शोणितेन मद्येन च विषेण च । षट्स्वप्येतासु पित्तं हि प्रभुत्वेनावतिष्ठते ॥ (Su.U.46/8)

Ācārya Caraka and Śārṅgadhara classified Mūrcchā into 4 types namely Vātaja, Pittaja, Kaphaja and Sannipātaja Mūrcchā. Whereas Ācārya Suśruta classified Mūrcchā into 6 types namely Vātaja, Pittaja, Kaphaja, Śoṇitaja, Madyaja and Viṣaja.

Vātaja Mūrcchā

- ⊙ नीलं वा यदि वा कृष्णमाकाशमथवाऽरुणम्। पश्यंस्तमः प्रविशति शीघ्रं च प्रतिबुध्यते ॥
  वेपथुश्चाङ्गमर्दश्च प्रपीडा हृदयस्य च। कार्श्यं श्यावारुणा च्छायामूर्च्छाये वातसम्भवे॥ (Ca.Sū.24/35-36)

The clinical features of Vātaja mūrcchā are Nīlaṃ vā kṛṣṇamākāśamathavāaruṇam paśyaṃstamaḥ praviśati (the person become unconscious after seeing the sky in blue, black or reddish colour), Śīghraṃ ca pratibudhyate (regains consciousness very quickly), Vepathu (tremors), Aṅgamarda (malaise), Prapīḍā hṛdayasya (pain in the cardiac region), Kārśyam (emaciation) and Śyāvāruṇā cchāyā (blackish and reddish complexion).

Pittaja Mūrcchā

- ⊙ रक्तं हरितवर्णं वा वियत् पीतमथापि वा। पश्यंस्तमः प्रविशति सस्वेदः प्रतिबुध्यते॥
  सपिपासः ससन्तापो रक्तपीताकुलेक्षणः। सम्भिन्नवर्चाः पीताभो मूर्च्छाये पित्तसम्भवे॥ (Ca.Sū.24/37-38)

The clinical features of Pittaja Mūrcchā are Raktaṃ Harita varṇaṃ vā pītam paśyaṃstamaḥ praviśati (the person become unconscious after seeing the sky in red, green or yellowish colour), Sasvedaḥ pratibudhyate (regains conscious with profuse perspiration), Sapipāsa (thirst), Sasantāpa (increased body temperature), Raktapitakulekṣaṇa (reddish or yellowish distressed eyes), Sambhinna varcāḥ (loose stools) and Pītābho (yellowish complexion).

Kaphaja Mūrcchā

- ⊙ मेघसङ्काशमाकाशमावृतं वा तमोघनैः। पश्यंस्तमः प्रविशति चिराच्च प्रतिबुध्यते॥
  गुरुभिः प्रावृतैरङ्गैर्यथैवार्द्रेण चर्मणा। सप्रसेकः सहृल्लासो मूर्च्छाये कफसम्भवे॥ (Ca.Sū.24/39-40)

The clinical features of Kaphaja Mūrcchā are Meghasaṅkāśamākāśamāvṛtaṃ vā tamoghanaiḥ paśyaṃstamaḥ praviśati (the person become unconscious after seeing the sky as if covered with clouds or covered with deep darkness), Cirācca pratibudhyate (regains conscious after a long time), Gurubhiḥ prāvṛtairaṅgairyathaivārdreṇa carmaṇā (feels heavy as if the body is wrapped with wet leather), Sapraseka (salivation) and Sahṛllāsā (nausea).

Sannipātaja Mūrcchā

- ⊙ सर्वाकृतिः सन्निपातादपस्मार इवागतः। स जन्तुं पातयत्याशु विना बीभत्सचेष्टितैः॥ (Ca.Sū.24/41)

The clinical features of Sannipātaja Mūrcchā are characterized by combination of all the three doṣas and Apasmāra ivāgata sajantuṃ pātayatyāśu vinā bībhatsaceṣṭitaiḥ (the person falls suddenly without abnormal body movements and regains his senses after a long period as seen in Apasmāra).

Raktaja Mūrcchā

- ⊙ पृथिव्यम्भस्तमोरूपं रक्तगन्धश्च तन्मयः ॥ तस्माद्रक्तस्य गन्धेन मूर्च्छन्ति भुवि मानवाः ।
  द्रव्यस्वभाव इत्येके दृष्ट्वा यदभिमुह्यति ॥ (Su.U.46/9-10)

Pṛthvi (earth) and Ambha (water) have predominance of Tamoguṇa; even the Raktagandha (smell of blood) contains the same attribute, i.e. Tamoguṇa. Therefore, the person becomes unconscious by the smell of blood. However, some become unconscious after seeing the blood itself due to the Dravya svabhāva (innate nature) of blood.

- ⊙ स्तब्धाङ्गदृष्टिस्त्वसृजा गूढोच्छ्वासश्च मूर्च्छितः । (Su.U.46/12)

The clinical features of Raktaja Mūrcchā are Stabdhāṅga (stiffness of the body) and Stabdha dṛṣṭi (fixed gaze) and Gūḍhocchvāsa (respiration becomes feeble).

Madyaja and Viṣaja Mūrcchā

- ⊙ गुणास्तीव्रतरत्वेन स्थितास्तु विषमद्ययोः । त एव तस्माज्जायेत मोहस्ताभ्यां यथेरितः ॥ (Su.U.46/11)

Madya (alcohol) and Viṣa (poison) has the same properties. Madya (alcohol) properties are slightly less potent when compared to Viṣa (poison); hence the clinical features are accordingly severe in Viṣa (poison) and moderate in Madya (alcohol).

- ⊙ मद्येन विलपंश्छेते नष्टविभ्रान्तमानसः । गात्राणि विक्षिपन् भूमौ जरां यावन्न याति तत् ॥ (Su.U.46/12)

The clinical features of **Madyaja Mūrcchā** are Vilapaṃśchete (delirious talks), Naṣṭa vibhrānta mānasaḥ (mental aberration) and Gātrāṇi vikṣipan bhūmau jarāṃ yāvanna yāti (falls unconscious with stretched out body parts and regains after the digestion of consumed Madya)

- ⊙ वेपथुस्वप्नतृष्णाः स्युः स्तम्भश्च विषमूर्च्छिते । वेदितव्यं तीव्रतरं यथास्वं विषलक्षणैः ॥ (Su.U.46/13)

The clinical features of **Viṣaja Mūrcchā** are Vepathu (tremors), Svapna (sleep), Tṛṣṇā (thirst) and Stambha (stiffness) associated with specific symptoms of respective poisons. By their symptoms, their Tīvrataraṃ (intensity) should be known.

SANNYĀSA

◉ दोषेषु मदमूर्च्छायाः कृतवेगेषु देहिनाम्। स्वयमेवोपशाम्यन्ति सन्न्यासो नौषधैर्विना॥
(Ca.Sū.24/42) / (A.H.Ni.6/36)

Mada and Mūrcchā disappear on their own as soon as the vitiated doṣas subside. However, Sannyāsa does not subside without the use of therapeutics.

◉ वाग्देहमनसां चेष्टामाक्षिप्यातिबला मलाः। सन्न्यस्यन्त्यबलं जन्तुं प्राणायतनसंश्रिताः॥
स ना सन्न्याससन्न्यस्तः काष्ठीभूतो मृतोपमः। प्राणैर्वियुज्यते शीघ्रं मुक्त्वा सद्यःफलाः क्रियाः॥ (Ca.Sū.24/43-44)

◉ वाग्देहमनसां चेष्टामाक्षिप्यातिबला मलाः। सन्न्यासं सन्निपतिताः प्राणायतनसंश्रयाः॥
कुर्वन्ति, तेन पुरुषः काष्ठीभूतो मृतोपमः। म्रियेत शीघ्रं शीघ्रं चेच्चिकित्सा न प्रयुज्यते॥ (A.H.Ni.6/37-38)

The aggravated doṣas gets located in the Praṇāyatana (heart and brain etc.) of weak person and afflicts his Vāk (speech) and Dehamanasāṃ ceṣṭa (physical and mental activities) and produces the disease Sannyāsa which is characterized by non responsive falls like Kāṣṭhībhūto (wooden log) and Mṛtopama (seems as if dead). If the condition is not treated quickly with appropriate treatment, it may turn fatal.

◉ प्रभूतदोषस्तमसोऽतिरेकात् सम्मूर्च्छितो नैव विबुध्यते यः ॥ सन्न्यस्तसञ्ज्ञो भृशदुश्चिकित्स्यो ज्ञेयस्तदा बुद्धिमता मनुष्यः।
(Su.U.46/20-21)

When the person becomes unconscious due to excessive aggravated doṣas and predominance of Tamas and does not regain consciousness, the physician should diagnose this condition as Sannyāsa which is very difficult to cure.

Ācārya Caraka and Suśruta explains the importance of immediate treatment in Sannyāsa with the simile that the lump of clay sinking in water should be retrieved before it dissolves, similarly in Sannyāsa; the person should be restored quickly before the sweat appears.

Sādhyāsādhyata of Sannyāsa

In case of Sannyāsa, if the person develops Ānāha (distention of abdomen), Lālā srāva (salivation) and Śvāsana (dyspnoea) along with Na labda saṃjña (does not regain consciousness) even after all the measures, then he should be discarded from the treatment.

In Contemporary science

Mūrcchā (fainting) and Sannyāsa (coma) are the progressive conditions of the Raktaja duṣṭi and it can be seen as prodromal symptom, symptom and complication of different diseases such as syncope, respiratory conditions such as Kussmoull's breathing, Cheyne tokes breathing etc., metabolic diseases such as hypertension and diabetes mellitus etc.

MCQ's

1. Pralaya is the synonym of

a) Mada b) Mūrcchā
c) Sannyāsa d) Both B and C

2. According to Caraka, Mūrcchā is of .... types

a) 3 b) 4
c) 5 d) 6

3. Stabdha Dṛṣṭi is the feature of

a) Vātaja Mūrcchā b) Pittaja Mūrcchā
c) Raktaja Mūrcchā d) Viṣaja Mūrcchā

4. Which Srotas can leads to Mūrcchā

a) Rasavaha b) Raktavaha
c) Saṃjñavaha d) All of the above

5. Meghasaṅkāśamākāśamāvṛtaṃ vā tamoghanaiḥ is seen in

a) Vātaja Mūrcchā c) Pittaja Mūrcchā
b) Kaphaja Mūrcchā d) Raktaja Mūrcchā

6. Which of the following will subside without Auṣadha

a) Mada b) Mūrcchā
c) Sannyāsa d) Both A and B

Answer Keys

1	b	2	b	3	c	4	d	5	c	6	d

◆

TOPIC 53

Common Psychiatric Diseases

Introduction

Psychiatric diseases, or mental health disorders, encompass a broad spectrum of conditions that profoundly impact an individual's mood, cognition, and conduct. These conditions can significantly impede a person's ability to navigate daily life, sustain relationships, and manage stress. Major psychiatric illnesses include Anxiety disorders such as generalized anxiety disorder and panic disorder; Mood disorders such as depression and bipolar disorder; Psychotic disorders such as schizophrenia; Personality disorders such as borderline personality disorder; Trauma and Stress related disorders such as post-traumatic stress disorder (PTSD); Neuro-developmental disorders such as attention deficit hyperactivity disorder (ADHD); Substance-Related and Addictive disorders such as substance abuse and dependence; Eating disorders such as anorexia nervosa and bulimia nervosa; Sleep-Wake disorders such as insomnia and sleep apnoea; and other mental health conditions like obsessive-compulsive disorder (OCD), dissociative disorders, and somatic symptom disorder.

DEPRESSION

Introduction

Depression is a mood disorder and it is characterized by persistent feeling of sadness nd loss of interest. The prevalence of depression is higher in females during their early dolescence period and after the age of 50-60 years; the incidence is equal in both genders. research study conducted in 2017 shows that 45.7 million people had depressive disorders India.

The exact causes of depressive disorders are not fully understood. But genetic factors, environmental factors and neuro chemical abnormalities play a major role. Genetic factors show possible genetic predisposition and first-degree relatives of depressed individuals as likely to develop depression. Environmental factors such as death of a spouse, lack or reduced social support, financial problems, interpersonal difficulties and assault on self esteem etc., are bearing on the development of depression. Neurotransmitters like nor-epinephrine, serotonin, dopamine, glutamate and brain-derived neurotrophic factor play an important role in causing depression.

Classification:

The Diagnostic and Statistical Manual for Mental Disorders (DSM-V) has classified depressive disorders into disruptive mood dysregulation disorder, major depressive disorder, persistent depressive disorder (dysthymia), premenstrual dysphoric disorder, substance/medication-induced depressive disorder, depressive disorder due to another medical condition, other specified depressive disorder and unspecified depressive disorder.

- **Disruptive mood dysregulation disorder:** is a childhood depressive disorder under 12 years of age and is characterised by persistent irritability and frequent episodes of extreme behavioral dyscontrol.
- **Major depressive disorder:** is the most common depressive disorder and is characterized by persistently low or depressed mood, anhedonia (decreased interest) in pleasurable activities, feelings of guilt or worthlessness, lack of energy, poor concentration, appetite changes, psychomotor retardation or agitation, sleep disturbances, or suicidal thoughts.
- **Persistent depressive disorder (dysthymia):** is a chronic and persistent depressive disorder characterized by depressed mood that continues for at least 2 years in adults or 1 year in children.
- **Premenstrual dysphoric disorder:** is characterized by dysphoria, mood lability, irritability, and anxiety that occur repeatedly during the premenstrual phase of the cycle and resolve around the time of (or after) menses.
- **Substance/medication-induced depressive disorder:** is characterized by prominent symptoms of depression as a result of substance intoxication or withdrawal or to a medication treatment.
- **Depressive disorder due to another medical condition:** is characterized by prominent symptoms of depression as a result of physiological consequence of another medical condition.

Clinical features:

The common features of these disorders are sadness, emptiness, or irritable mood accompanied by sleep disturbance, interest/pleasure reduction, guilt feelings or thoughts of worthlessness, energy changes/fatigue, concentration/attention impairment, appetite/weight changes, psychomotor disturbances, suicidal thoughts and depressed mood.

Diagnosis:

Diagnosis is made on the basis of clinical features and DSM-V criteria. Depression has to be differentiated from organic disorders, schizophrenia, endocrine disorders and substance use disorders.

ANXIETY NEUROSIS

Introduction

Anxiety neurosis, also known as anxiety disorder, is a group of mental health conditions characterized by overwhelming and uncontrollable feelings of anxiety and fear. These feelings significantly impair a person's social, occupational, and personal functioning. Globally, anxiety disorders are the most prevalent mental health conditions, affecting both males and females, including children. Notably, the prevalence is higher among females than males. According to a 2017 research study, approximately 44.9 million people in India suffered from anxiety disorders.

The causes of anxiety disorders are not fully understood. But various theories like biological, psychological and social factors are involved in manifesting the disease.

- **Biological causes** may include hereditary, neurotransmitter imbalance especially nor epinephrine, serotonin and gamma aminobutyric acid (GABA), physical disorders like malignancy, diabetes, hyperthyroidism, pheochromocytoma, etc., medications such as corticosteroids, cocaine, amphetamines, caffeine etc.
- **Psychological causes** may include personality traits, stress, low self-esteem, cognitive conflicts, negative emotions, poor coping strategies, inter or intra personal conflicts, developmental crisis and perception of situational crisis etc.
- **Social causes** may include ending of a significant relationship, exposure to a life-threatening disaster, child abuse and substance abuse

Classification:

The Diagnostic and Statistical Manual for Mental Disorders (DSM-V) has classified anxiety disorders into separation anxiety disorder, selective mutism, specific phobias, social anxiety isorder (SAD), panic disorder, agoraphobia, generalised anxiety disorders (GAD), obsessive ompulsive disorder (OCD) Substance/medication-induced anxiety disorder and anxiety isorder due to another medical condition

- **Separation anxiety disorder:** is one of the most common childhood anxiety disorders and refers to excessive anxiety or distress for the developmental level and is related to separation from attachment figures (someone or something that provides the individual with comfort). Although the symptoms often develop in childhood, they can manifest throughout adulthood.
- **Selective mutism:** is characterized by a persistent failure to speak in specific social situations in which there is an expectation to speak (e.g., school), even though the individual speaks in other situations.
- **Specific phobias (simple phobia):** is characterized by clinically significant anxiety provoked by exposure to a specific feared object or situation such as a fear of certain animals (especially snakes, rodents and dogs), insects, lightning, heights, flying, blood or injections etc., often leading to avoidance behaviour.
- **Social anxiety disorder (social phobia):** is characterized by clinically significant anxiety provoked by exposure to certain types of social or performance situations, often leading to avoidance of social interaction.
- **Panic disorder:** is characterized by recurrent unexpected panic attacks and persistent concern about additional attacks or their consequences. Panic attack is a discrete period of intense fear and discomfort in the absence of real danger or associated with feelings of impending doom that is accompanied by symptoms like palpitations, trembling, sensation of shortness of breath, feeling of choking, chest pain or discomfort, fear of losing control, fear of dying.
- **Agoraphobia:** is characterized by specific anxiety regarding places or situations from which escape might be difficult in the event of a panic attack or panic symptoms.
- **Generalised anxiety disorders (GAD):** is characterized by persistent and excessive anxiety and worry about a number of events or activities often for a period of at least 6 months.
- **Obsessive compulsive disorder (OCD):** is characterized by recurrent and intrusive thoughts (called obsessions) and repetitive behaviours that the person feels driven to perform (called compulsions) that cause marked distress or anxiety.
- **Substance/medication-induced anxiety disorder:** is characterized by prominent symptoms of anxiety as a result of substance intoxication or withdrawal or to a medication treatment.
- **Anxiety disorder due to another medical condition:** is characterized by prominent symptoms of anxiety as a result of physiological consequence of another medical condition.

Clinical features:

The physical features of anxiety disorder are restlessness, tremors, muscle tension, headache, dizziness, shortness of breath, hyperventilation, paresthesia (tingling or pricking sensation), difficulty swallowing, flushing, pallor, tachycardia, palpitations, sweating, cold and clammy hands, diarrhoea, dry mouth and urinary frequency; the psychological symptoms of anxiety disorder are severe anxiety, worry, irritability, insomnia, feeling of dread, decreased libido and lack of concentration or attention

Diagnosis:

Diagnosis is based on the clinical history and DSM-V. Clinical history helps to rule out the underlying medical conditions such as diabetes, hypo or hyperthyroidism etc., and medications such as alcohol, tobacco, cannabis, sedatives, opioids etc., causing anxiety disorders. DSM-V criteria helps to identify the specific type of anxiety disorder causing symptoms as well as any other possible disorders that may be involved.

EPILEPSY (NON-ORGANIC)

Introduction

Non-organic epilepsy, also known as functional or psychogenic epilepsy, pseudoseizures, dissociative seizures, or Psychogenic Non-Epileptic Seizures (PNES), refers to seizures that are not caused by abnormal brain electrical activity, but rather by psychological or emotional factors such as trauma, stress or emotional conflicts. Globally, it is estimated that 2-33% of epilepsy diagnoses are Psychogenic Non-Epileptic Seizures, with 10-20% of epilepsy diagnoses in India being Psychogenic Non-Epileptic Seizures. This condition is most prevalent among young adults aged 15 to 35, particularly females.

Pathophysiology:

The precise pathophysiology of PNES is not fully understood, but it is believed to involve a complex network disorder affecting several brain regions responsible for sensorimotor rocessing, emotional regulation, and stress responses. Functional neuroimaging studies have hown abnormalities in limbic structures such as the amygdala (fear and anxiety processing), ippocampus (memory formation and consolidation), temporal lobe (memory, emotion egulation), and prefrontal cortex (executive function, decision-making). These seizures are a rm of conversion disorder, where psychological stress is converted into physical symptoms. tients with PNES do not have conscious control over their seizures, and the condition is en misdiagnosed as epilepsy.

nical features:

Psychogenic seizures, which are non-epileptic in nature, bear resemblance to epileptic ures and are often indicative of a conversion reaction triggered by underlying psychological ess. Specific manifestations such as side-to-side turning of the head, ictal eye closure, asymmetrical and high-amplitude limb movements, twitching of all four extremities without loss of consciousness, and pelvic thrusting are more frequently associated with psychogenic seizures rather than epileptic seizures. Moreover, psychogenic seizures typically endure for longer durations than epileptic seizures and may exhibit fluctuations over periods ranging from minutes to hours.

Diagnosis:

Diagnosis is based on the analysis of video EEG (electroencephalogram) and the criteria outlined in the DSM-5. Notably, video EEG recordings reveal normal electrical brain activity during Psychogenic Non-Epileptic Seizures. As Psychogenic Non-Epileptic Seizures is classified as a conversion disorder, as per the DSM-5, the diagnostic criteria encompass the following key elements.

- **Symptoms:** One or more symptoms of altered voluntary motor or sensory function.
- **Incompatibility:** Clinical findings provide evidence of incompatibility between the symptom and recognized neurological or medical conditions.
- **Exclusion:** The symptom or deficit is not better explained by another medical or mental disorder.
- **Distress or Impairment:** The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation.
- **Specific Symptom Type:** The specific symptom type must be reported "with attacks or seizures."

MCQ's

1. Which of the following neurotransmitters is NOT mentioned as playing a role in causing depression

a) Norepinephrine b) Serotonin

c) Dopamine d) Acetylcholine

2. Diagnosis of depression is made on the basis of clinical features and which criteria

a) ICD-10 b) DSM-V

c) WHO guidelines d) APA guidelines

3. Depression must be differentiated from which of the following disorders

a) Organic disorders b) Schizophrenia

c) Endocrine disorders d) All of the above

4. Which neurotransmitter is NOT mentioned as playing a role in anxiety disorders
   a) Norepinephrine b) Serotonin
   c) Dopamine d) Gamma aminobutyric acid (GABA)
5. Which anxiety disorder is characterized by a persistent failure to speak in specific social situations despite speaking in other situations
   a) Separation anxiety disorder b) Selective mutism
   c) Social anxiety disorder d) Panic disorder
6. Which of the following is NOT classified as a depressive disorder according to the DSM-V
   a) Major depressive disorder
   b) Bipolar disorder
   c) Persistent depressive disorder
   d) Substance/medication-induced depressive disorder
7. Which of the following is NOT a symptom of major depressive disorder
   a) Anhedonia b) Feelings of guilt or worthlessness
   c) Increased energy levels d) Suicidal thoughts
8. Which of the following is NOT a common clinical feature of Psychogenic Non-Epileptic Seizures (PNES)Tonic seizure
   a) Side-to-side turning of the head
   b) Ictal eye closure
   c) Symmetrical and rhythmic limb movements
   d) Pelvic thrusting
9. According to the DSM-5, which of the following is NOT a diagnostic criterion for Psychogenic Non-Epileptic Seizures (PNES)
   a) One or more symptoms of altered voluntary motor or sensory function
   b) Clinical findings provide evidence of incompatibility between the symptom and recognized neurological or medical conditions
   c) The symptom or deficit is better explained by another medical or mental disorder
   d) The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning

Answer Keys

1	d	2	b	3	d	4	c	5	b	6	b	7	c	8	c	9	c

TOPIC 54

Phiraṅga and Upadaṃśa

PHIRAṄGA ROGA

Phiraṅga roga is a Āgantuja roga (exogenous disease), which spreads to others through physical and sexual contact with the person who belongs to Phiraṅga deśa. Hence it is named as Phiraṅga roga. Gandha Roga (Bhāvaprakāśa) and Priyāṅga Roga (Arka prakāśa) are the synonyms of Phiraṅga roga and the disease spreaded among Indians through the people of Phiraṅga deśa (Portugal) around 14th century A.D., hence there is no description available in Bṛhatrayi's and Ācārya Bhāvamiśra (16th century A.D.) is the first person, who explained this disease in detail in his book Bhāvaprakāśa. Ācārya Gananath Sen explained two types of Phiraṅga roga i.e. Svakṛta (acquired) and Janmadoṣakṛta (congenital). In Janmadoṣakṛta (congenital), the disease spreads to child from mother through Mātṛrakte (blood from mother). It is also called as Bījadoṣakṛta Phiraṅga by some scholars.

Nirūkti and Paribhāṣa

- ⦿ फिरङ्गसंज्ञके देशे बाहुल्येनैव यद्भवेत् । तस्मात्फिरङ्ग् इत्युक्तो व्याधिर्व्याधिविशारदैः ॥ (B.P.Ci.59/1)

The disease is more predominant in Phiraṅga deśa (Portugal) hence it is named as Phiraṅga roga.

- ⦿ फिरङ्गजुष्टस्त्रीपुंस संयोगात् कालतोगात् । क्षतं यत् कठिनस्पर्शं शिश्नादौ मण्डलाकृति ॥ (Si.Ni.10/687)

Due to Strīpuṃsa saṃyogāt (copulation by men and women), the disease Phiraṅga roga develops after some time in the form of Kṣata (ulcer) develops on the Śiśna (penis) which are Kaṭhinasparśa (hard to touch) and Maṇḍalākṛti (round in shape).

Nidāna and Saṃprāpti

◉ गन्धरोगः फिरङ्गऽयं जायते देहिनां ध्रुवम् । फिरङ्गिणोऽङ्गसंसर्गात्फिरङ्गिण्याः प्रसङ्गतः ।
व्याधिरागन्तुजो ह्येष दोषाणामत्र संक्रमः । भवेत्तं लक्ष्येत्तेषां लक्षणैर्भिषजां वरः ॥ (B.P.Ci.59/2-3)

Phiraṅga roga is also known as Gandharoga i.e. highly contagious condition, which occurs due to Phiraṅgiṇo aṅgasaṃsargāt (physical contact with diseased female belonging to Phiraṅga deśa) and Phiraṅgiṇyā prasaṅgataḥ (sexual contact with diseased female belonging to Phiraṅga deśa). As it is a Āgantuja roga (exogenous disease), the involvement of doṣas takes place later. Based on the manifestation of Lakṣanās (clinical features) the involvement of doṣas should be assessed.

Saṃprāpti Ghaṭaka of Phiraṅga roga

Doṣa	Pittakapha
Dūṣya	Rakta, Māṃsa, Meda, and Asthi
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rakta, Māṃsa, Meda, and Asthi
Srotoduṣṭi	Saṅga and Atipravṛtti
Sañcāra sthāna	Sarva Śārīra
Adhiṣṭhāna	Śiśna and Sarva Śārīra
Udbhava sthāna	Āmapakvāśaya
Vyakta sthāna	Śiśna and Sarva Śārīra
Rogamārga	Bāhyābhyantara

Bheda and Lakṣanās of Phiraṅga roga

◉ फिरङ्गस्त्रिविधो ज्ञेयो बाह्यश्चाभ्यन्तरस्तथा । बहिरन्तर्भवश्चापि तेषां लिङ्गानि च ब्रुवे ॥ (B.P.Ci.59/4)

Phiraṅga roga is classified into three types namely Bāhya (external), Ābhyantara (internal) and Bahirantarbhava (external and internal).

◉ तत्र बाह्यः फिरङ्गः स्याद्विस्फोटसदृशोऽल्परुक् । स्फुटितो व्रणवद्वेद्यः सुखसाध्योऽपि स स्मृतः ॥
सन्धिष्वाभ्यन्तरः स स्यादामवात इव व्यथाम् । शोथञ्च जनयेदेष कष्टसाध्यो बुधैः स्मृतः ॥ (B.P.Ci.59/5-6)

Visphoṭa sadṛśa (lesion manifested on the penis resembles the blisters), Alparuk (mild pain) and Sphuṭito vraṇavatvedyaḥ (ulcer is formed on rupture of lesions) are the clinical features of Bāhya (external) Phiraṅga roga. And it is Sukha sādhya (easily curable).

Ābhyantara (internal) Phiraṅga roga affects the Sandhi (joints) and produces Vyathām (pain) similar to Āmavāta associated with Śotha (swelling). And it is Kaṣṭa sādhya (difficult to cure).

The clinical features of both Bāhya (external) and Ābhyantara (internal) Phiraṅga roga are manifested in Bahirantarbhava (external and internal) Phiraṅga roga.

Upadravā of Phiraṅga roga

◉ कार्श्यं वलक्षयो नासभङ्गो वह्नेश्च मन्दता । अस्थिशोषोऽस्थिवक्रत्वं फिरङ्गोपद्रवा अमी ॥ (B.P.Ci.59/7)

Kārśya (weakness), Balakṣaya (loss of strength), Nāsabhaṅga (saddle nose or depressed bridge of nose), Vahneśca mandatā (diminution of digestive fire), Asthiśoṣa (desiccation or atrophy of the bones) and Asthivakratva (curvature or deformity of bones) are the complications of Phiraṅga roga.

Sādhyāsādhyata of Phiraṅga roga

◉ बहिर्भवो भवेत्साध्यो नवीनो निरुपद्रवः । आभ्यन्तरस्तु कष्टेन साध्यः स्यादयमामयः ॥
बहिरन्तर्भवा जीर्णः क्षीणस्योपद्रवैर्युतः । व्याप्तो व्याधिरसाध्योऽयमित्याहुर्मुनयः पुराः ॥ (B.P.Ci.59/8-9)

Bāhya (external) Phiraṅga, which is Navīna (recent onset) and Nirupadrava (without complications) is Sādhya (easily curable), whereas Ābhyantara (internal) Phiraṅga is Kaṣṭa sādhya (difficult to cure). Bahirantarbhava (external and internal) Phiraṅga, which is Jīrṇa (chronic), Kṣīṇā (afflicts the weak person), Upadravairyuta (with complications) and Vyāpto vyādhi (spreads to all over the body) is Asādhya (incurable).

In Contemporary Science

Phiraṅga roga can be correlated to syphilis in Contemporary science. (Please refer Topic 55)

UPADAṂŚA

Upadaṃśa is a disease which afflicts the male genitals mainly through sexual contact. Ācārya Suśruta described the disease Upadaṃśa after Vṛddhi because of the similarity in the causative factors such as Atimaithuna (excessive coitus) etc. Ācārya Vāgbhaṭa explained Upadaṃśa along with Śūka rogas in the chapter Guhyaroga vijñānīyaṃ. Āḍhamalla described that the term 'Guhya' symbolizes that the disease may also manifest in females. Ḍalhaṇa and Yogaratnākara state that this dreadful disease occurs even in females.

Nirūkti and Paribhāṣā

◉ उपस्थरोगभेदे उपदंश । (Vācaspatyam)

◉ मेढ्ररोगविशेषः । (SKD)

Upadaṃśa is defined as a disease afflicting the genitals (Upastha roga bheda) especially affecting the male genital i.e. Penis (Meḍhra roga Viśeṣa).

◉ उपदंशसंज्ञा रूढा बोद्धव्या, व्रणविशेषेन मेढ्रे लिङ्गे पञ्चप्रकारो भवति ॥ (Āḍ.Śā.P.7/82)

Appearance of Rūḍhā (sprouts or growth) especially formation of ulcer in the Meḍhra (penis) is known as Upadaṃśa which is of five types.

◉ उपदंशसञ्ज्ञा च दंशनोपाधिमन्तरेणापि रूढा बोद्धव्या। (Ma.M.N.47/1)

Appearance of Rūḍhā (sprouts or growth) after a Daṃśana (bite) in the Meḍhra (penis) is known as Upadaṃśa.

◉ क्षतेऽक्षते वा श्वयथुमुपजनयन्ति, तमुपदंशमित्याचक्षते । (Su.Ni.12/7)

Appearance of swelling of the penis (Śvayathu means Śiśnagata śopha) with or without a wound is known as Upadaṃśa.

◉ उपस्थक्षतकृ द्व्याधिरुपदंश इतीर्यते ॥ (Si.Ni.10/675)

The disease which produces Kṣata (injury) on the Upastha (penis) is called Upadaṃśa.

Sāmānya Nidāna and Saṃprāpti of Upadaṃśa

◉ हस्ताभिघातान्नखदन्तपातादधावनाद्रत्यतिसेवनाद्वा । योनिप्रदोषाच्च भवन्ति शिश्ने पञ्चोपदंशा विविधापचारैः ॥ (M.N.47/1) / (B.P.M.51/1)

Due to Hastābhighāta (Hastena maithunāt – excessive masturbation), Nakhadantāghāt (injury to the genitals with nails and teeth), Adhāvanāt (not cleaning the genitals i.e. unhygienic), Atyupasevanāt (excessive sexual intercourse), Yonipradoṣāt (sexual contact with diseased vagina), Vividhāpacāra (Aśuddhasalila prakṣālana – washing the genitals with unclean water) are the causative factors of the disease Upadaṃśa which is of five types.

◉ तत्रातिमैथुनादतिब्रह्मचर्याद्वा तथाऽतिब्रह्मचारिणीं चिरोत्सृष्टां रजस्वलां दीर्घरोमां कर्कशरोमां सङ्कीर्णरोमां निगूढरोमामल्पद्वारां महाद्वारामप्रियामकामामचौक्षसलिलप्रक्षालितयोनिमप्रक्षालितयोनिं योनिरोगोपसृष्टां स्वभावतो वा दुष्टयोनिं वियोनिं वा नारीमत्यर्थमुपसेवमानस्य तथा करजदशनविषशूकनिपातनाद्बन्धनाद्धस्ताभिघाताच्चतुष्पदीगमनाद चौक्षसलिलप्रक्षालनादवपीडनाच्छुक्रवेगविधारणान्मैथुनान्ते वाऽप्रक्षालनादिभिर्मेढ्रमागम्य प्रकुपिता दोषाः क्षतेऽक्षते वा श्वयथुमुपजनयन्ति, तमुपदंशमित्याचक्षते ॥ (Su.Ni.12/7)

Due to Atimaithunāt (excessive sexual intercourse) or Atibrahmacaryāt (prolonged celibacy), Atibrahmacāruṇiṃ (intercourse with celibate woman), Cirotsṛṣṭāṃ (Prabhūtakālatyaktāṃ - intercourse with the abandoned abundant period for sex), Rajasvālāṃ (intercourse with menstruating woman), Dīrgharomāṃ Karkaṣaromāṃ saṅkīrṇaromāṃ nigūḍharomāṃ (intercourse with woman with long, rough, thick and concealed pubic hair), Alpadvāraṃmahādvāram (intercourse with narrow and wide vaginal openings), Apriyā (Apriyāyāṃ liṅgānuthānenetaścetābhighāta prasaṅgāt - intercourse with unfriendly woman leads to injury to the penis and erectile dysfunction), Akāmā (Sakthisankocena dvārālpatvāt - intercourse with unwilling woman leads to contraction of vaginal opening and thighs) Acaukṣa (Apavitram - intercourse with impure or unclean woman), Salilaprakṣālita yonimaprakṣālitayoniṃ (intercourse with woman who has cleaned genitals with unclean water or not cleaned the genitals), Yonirogopasṛṣṭāṃ (intercourse with woman who suffers from genital disease), Svabhāvato vā duṣṭayoniṃ (intercourse with woman who has distorted vagina naturally or defective vagina), Viyoniṃ (intercourse with woman in any of the natural fissures of her body other than vagina), Nārimatyarthamupasevamānasya tathākarajadaśana viṣaśūkanipātanād bandhanāddhastābhighātāt (excessive intercourse with woman and injury of the genitals with finger nails, teeth, poison, poisoned water insect, squeezing, bandaging or by masturbation), Catuṣpadīgamanāt (intercourse with female quadrupeds), Acaukṣhasalilaprakṣālanāt (washing the genitals with unclean water), Avapīḍanāt (by pressing the genitals), Śukravegavidhārāṇānmaithunānte (suppressing the semen at the end of intercourse) or Aprakṣālanādibhimeḍhramāgamya (not cleaning the genitals after the intercourse) are the causative factors of Upadaṃśa.

◉ स्त्रीव्यवायनिवृत्तस्य सहसा भजतोऽथवा। दोषाध्युषितसङ्कीर्णमलिनाणुरजः पथाम्॥
अन्ययोनिमनिच्छन्तीमगम्यां नवसूतिकाम्। दूषितं स्पृशतस्तोयं रतान्तेष्वपि नैव वा॥
विवर्धयिषया तीक्ष्णान् प्रलेपादीन् प्रयच्छतः। मुष्टिदन्तनखोत्पीडाविषवच्छूकपातनैः॥
वेगनिग्रहदीर्घातिखरस्पर्शविघट्टनैः। दोषा दुष्टा गता गुह्यं त्रयोविंशतिमामयान्॥
जनयन्त्युपदंशादीन् ....॥ (A.H.U.33/1-4)

Ācārya Vāgbhaṭa added Doṣādhyuṣita (intercourse with woman whose vaginal passage is vitiated with doṣas) and Navasūtikām (intercourse with woman who recently delivered) as causative factors. These lead to aggravation of doṣas and it gets localized in the penis resulting in Śiśnagata śopha (inflammatory swelling of the penis) with or without a wound which is known as Upadaṃśa.

Saṃprāpti Ghaṭaka of Upadaṃśa

Doṣa	Pitta Pradhāna Tridoṣa
Dūṣya	Rakta, Māṃsa and Śukra / Ārtava
Agni	Jaṭharāgni māndhya
Āma	Jaṭharāgni māndhya janya Āma
Srotas	Rakta, Māṃsa, Mūtra and Śukra vaha

Srotoduṣṭi	Saṅga and Atipravṛtti
Sañcāra sthāna	Mūtra and Śukra vaha
Adhiṣṭhāna	Śiśna
Udbhava sthāna	Āmapakvāśaya
Vyakta sthāna	Śiśna
Rogamārga	Bāhyābhyantara

Bheda and Lakṣaṇās of Upadaṃśa

- स पञ्चविधस्त्रिभिर्दोषैः पृथक् समस्तैरसृजा चेति । (Su.Ni.12/8)
- .....उपदंशोऽत्र पञ्चधा। पृथग्दोषैः सरुधिरैः समस्तैश्च.... । (A.H.U.33/5)

Upadaṃśa is classified into five types namely Vātaja, Pittaja, Kaphaja, Sannipātaja and Raktaja.

Vātaja Upadaṃśa

- तत्र वातिके पारुष्यं त्वक्परिपुटनं स्तब्धमेढ्रता परुषशोफता विविधाश्च वातवेदनाः । (Su.Ni.12/9)
- सतोदभेदैः स्फुरणैः सकृष्णैः स्फोटैर्व्यवस्येत् पवनोपदंशम् । (M.N.47/2)
- ....अत्र मारुतात्॥ मेढ्रे शोफो रुजश्चित्राः स्तम्भस्त्वक्परिपोटनम्। (A.H.U.33/5-6)

Pāruṣya (roughness), Tvakpariputaṇaṃ (cracking of the skin), Stabdhameḍhratā (penile rigidity), Paruṣa śophatā (hard swelling of the genitals), Vividha Vātavedanā (different types of Vātika pains i.e. Toda (pricking pain), Bheda (cutting pain) and Sphuraṇa (throbbing pain)) and Sakṛṣṇa sphoṭa (black papules) are the clinical features of Vātaja Upadaṃśa.

Pittaja Upadaṃśa

- पैत्तिके ज्वरः श्वयथुः पक्वोदुम्बरसङ्काशस्तीव्रदाहः क्षिप्रपाकः पित्तवेदनाश्च; (Su.Ni.12/9)
- पीतैर्बहुक्लेदयुतैः सदाहैः पित्तेन...॥ (M.N.47/2)
- पक्वोदुम्बरसङ्काशः पित्तेन श्वयथुर्ज्वरः॥ (A.H.U.33/6)

Jvara (fever), Śvayathu (swelling) which resembles Pakvodumbara (the colour of a ripe Indian fig fruit), Tīvra dāha (severe burning sensation), Kṣiprapāka (early suppuration), Pitta vedanā (Paittika type of pain) and Pītairbahukleda yukta (excessive yellowish discharge) are the clinical features of Pittaja Upadaṃśa.

Kaphaja Upadaṃśa

- श्लैष्मिके श्वयथुः कण्डूमान् कठिनः स्निग्धः श्लेष्मवेदनाश्च; (Su.Ni.12/9)
- सकण्डुरैः शोथयुतैर्महद्भिः शुक्लैर्घनैः स्रावयुतैः कफेन ॥ (M.N.47/3)
- श्लेष्मणा कठिनः स्निग्धः कण्डूमान् शीतलो गुरुः। (A.H.U.33/7)

Mahadbhi Kaṭhinaḥ Snigdhaḥ Śvayathu (big, hard and smooth swelling) of the genitals associated with Śitala (cold), Guru (heavy) and Kaṇḍu (itching sensation), Śuklaghanair srāva (thick whitish discharge) and Śleṣmavedanā (Ślaiṣmika type of pain) are the clinical features of Kaphaja Upadaṃśa.

Raktaja Upadaṃśa

- रक्तजे कृष्णस्फोटप्रादुर्भावोऽत्यर्थमसृक्प्रवृत्तिः पित्तलिङ्गान्यत्यर्थं ज्वरदाहौ शोषश्च, याप्यश्चैव कदाचित्; (Su.Ni.12/9)
- रक्तात् पिशितावभासैः ॥ स्फोटैः सकृष्णै रुधिरं स्रवन्तं रक्तात्मकं पित्तसमानलिङ्गम् । (M.N.47/2-3)
- शोणितेनासितस्फोटसम्भवोऽस्रस्रुतिर्ज्वरः॥ (A.H.U.33/7)

Piṣitāvabhāsa Kṛṣṇa sphoṭa (black papules which resemble the muscles) associated with Atyarthamasṛkpravṛtti (excessive bleeding) and Pittaliṅgānyartha (other clinical features of Pittaja Upadaṃśa) such as Jvara (fever), Dāha (burning sensation), Śoṣa (dryness) and Raktātmaka (reddish in colour) are the clinical features of Raktaja Upadaṃśa. It is Yāpya vyādhi (manageable disease).

Sannipātaja Upadaṃśa

- सर्वजे सर्वलिङ्गदर्शनमवदरणं च शेफसः कृमिप्रादुर्भावो मरणं चेति ॥ (Su.Ni.12/9)
- नानाविधस्रावरुजोपपन्नमसाध्यमाहुस्त्रिमलोपदंशम् । (M.N.47/4)
- सर्वजे सर्वलिङ्गत्वं श्वयथुर्मुष्कयोरपि। तीव्रा रुगाशुपचनं दरणं कृमिसम्भवः॥ (A.H.U.33/8)

All the clinical features of Upadaṃśa caused by three doṣas associated with Muṣkayo śvayathu (scrotal swelling), Āśupacana (early suppuration), Daraṇa ca śepha (cracking of penis), Kṛmiprādurbhāva (appearance of maggots), Nānāvidha srāva (different types of discharges) and Tīvra Ruja (severe pain) are the features of Sannipātaja Upadaṃśa and it leads to death.

Sādhyāsādhyata of Upadaṃśa

- याप्यो रक्तोद्भवस्तेषां मृत्यवे सन्निपातजः। (A.H.U.33/9)
- विशीर्णमांसं क्रिमिभिः प्रजग्धं मुष्कावशेषं परिवर्जयेच्च ॥ (M.N.47/4)

Raktaja Upadaṃśa is Yāpya (manageable) and Sannipātaja Upadaṃśa leads to Mṛtyu (death) and the following features of Upadaṃśa should be rejected for the treatment as it is Asādhya (incurable), they are Viśīrṇamāṃsa (destruction of muscles of the genitals), Krimibhiḥ prajagdhaṃ (Bhakṣitaṃ - maggots eating up the muscles) and Muṣkāvaśeṣa (entire muscles are destroyed and only the scrotum remains)

- सञ्जातमात्रे न करोति मूढः क्रियां नरो यो विषये प्रसक्तः । कालेन शोथक्रिमिदाहपाकैर्विशीर्णशिश्नो म्रियते स तेन। (M.N.47/5)

If the person indulges in sexual activity by ignoring the Upadaṃśa condition, then it leads to Śotha (swelling), Krimi (appearance of maggots), Dāha (burning sensation), Pāka (suppuration), Viṣīrṇa śiśna (destruction of the penis) and Mrīyate (death).

In Contemporary science,

Upadaṃśa can be correlated with Lymphogranuloma inguinale in Contemporary science. **Lymphogranuloma inguinale** is a sexually transmitted disease caused by the microorganism Chlamydia trachomatis serovars L1, L2 and L3. It is also called as Lymphogranuloma venereum or Nicolas-Favre disease. It is more common in tropical and sub-tropical climates. Research studies reported that the incidence of Lymphogranuloma inguinale ranges from 0.2 to 11.5% in India.

Incubation period: 3-30 days

Clinical features: Lymphogranuloma inguinale occurs in three stages i.e. primary, secondary and latent stage. In the primary stage, a small genital ulcer appears by three days after an exposure. Usually, it is symptomless and goes unnoticed by the patient. Rarely, the lesion gets more persistent and ulcerates extensively.

The secondary stage is commonly seen in males and begins within few days to several weeks after a primary lesion and it is characterized by swollen inflamed inguinal lymph nodes (buboes) which are firm, discrete and tender in the beginning. Later, the lymph nodes become matted together, and attached to the deeper tissues, causing inflammation of the overlying skin, which leads to multiple small abscesses and the formation of multiple sinuses that discharges pus or blood. Fever, chills, sweating, malaise, anorexia, arthralgia and myalgia are the constitutional features often preceded or associated with the onset of inflamed inguinal lymph nodes (buboes).

Latent stage begins as a sequel of secondary stage after a year or two to many years and is characterized by fistula, lymphatic obstruction, stricture and ulceration of the rectum and genital elephantiasis (chronic lymphatic edema produces sclerosing fibrosis of the subcutaneous tissue leading to induration and enlargement of scrotum and penis).

Diagnosis: Diagnosis is based on positive serology (complement fixation test, and microimmunofluorescence (micro-IF) test) or isolation of Chlamydia trachomatis from the infected site such as lymph nodes, urethra or rectum (by culture, nucleic acid amplification test (NAAT) or direct immunofluorescence).

MCQ's

1. Gandha roga is the synonym of
   a) Upadaṃśa b) Phiraṅga
   c) Pūyameha d) None of the above
2. Priyāṅga Roga is the synonym of
   a) Upadaṃśa b) Phiraṅga
   c) Pūyameha d) None of the above
3. Phiraṅga is of .... types
   a) 3 b) 4
   c) 5 d) 6
4. Āmavāta iva vyathām is seen in
   a) Bāhya Phiraṅga b) Ābhyantara Phiraṅga
   c) Both A and B d) None of the above
5. Which feature is not included in the Phiraṅga Upadrava
   a) Nāsa Bhaṅga b) Asthi Śoṣa
   c) Asthivakratva d) Asthi bheda
6. Upadaṃśa is of .... types
   a) 3 b) 4
   c) 5 d) 6
7. Pakvoḍambara saṅkāśa is seen in
   a) Vātaja Upadaṃśa b) Pittaja Upadaṃśa
   c) Kaphaja Upadaṃśa d) Sannipātaja Upadaṃśa
8. Kṛmiprādurbhāva is seen in
   a) Vātaja Upadaṃśa b) Pittaja Upadaṃśa
   c) Kaphaja Upadaṃśa d) Sannipātaja Upadaṃśa

9. Causative agent of Lymphogranuloma inguinale
   a) Chlamydia trachomatis
   b) Treponema pallidum
   c) Neisseria gonorrhoeae
   d) None of the above

10. Nicolas–favre disease is also known as
   a) Syphilis
   b) Lymphogranuloma inguinale
   c) Gonorrhoea
   d) None of the above

Answer Keys

1	b	2	b	3	a	4	b	5	d	6	c	7	b	8	d	9	a	10	b

◆

TOPIC 55

Syphilis and Gonorrhoea

Introduction

Sexually transmitted diseases or infections are commonly known as Venereal diseases that include variety of clinical syndromes caused by various pathogens which can be acquired and transmitted through sexual contact. Bacterial originated sexually transmitted diseases are Chlamydia, Syphilis, Gonorrhoea and Donovanosis; Viral originated sexually transmitted diseases are Herpes simplex, Genital warts and HIV; Fungal originated sexually transmitted diseases are Yeast infections (Candidiasis) and Parasitic originated sexually transmitted diseases are Trichomoniasis and Pubis lice.

SYPHILIS

Syphilis is a chronic inflammatory disease caused by spirochete bacteria Treponema pallidum and it may be congenital or acquired and is usually transmitted through sexual contact, blood transfusion, percutaneous injury and transplacental infection of the fetus may also occur.

Incubation period – is usually 3 weeks with a range from 9-90 days

Clinical features: The clinical features of syphilis may vary depending on the existing stages i.e. primary, secondary, latent and tertiary syphilis.

- **Primary syphilis** is characterized by single indurated painless papule called as primary chancre which develops 2- 3 weeks after exposure at the site of infection usually in genital area along with regional lymphadenopathy. The primary chancre heals within 4-6 weeks without treatment hence it passes unnoticed clinically.
- **Secondary syphilis** occurs 6-8 weeks after primary lesion and it is characterized by

wide spread diffuse or patchy mucocutaneous rash which is polymorphic (macular, papular and pustular) with little variation in size. Mostly rash is maculopapular, non itchy lesions distributed symmetrically on the trunk, proximal extremities, palms, soles, face and scalp. In certain moist areas of the body like perianal, inner aspects of thigh, lateral surface of scrotum, axillae, vulva and skin under the pendulous breasts, the papules may become fleshy looking masses due to constant moisture and rubbing called condyloma lata which are highly infectious. Other features include sore throat, fever, weight loss, malaise, headache, anorexia, arthralgia associated with generalized lymphadenopathy.

- **Latent syphilis** develops after a variable period ranging from 3-12 months after the primary and secondary stages. It may be early or late latent phases. Early latent phase is infectious phase with recurrent secondary infection and is defined as less than two years after infection and after the end of two years of infection, the person enters in to the late latent phase which is non infectious and the disease may persist in latent form for many years without treatment.
- **Tertiary syphilis** may develop 3 to 10 years after the primary stage and it is characterized by chronic localized granulomatus lesion called as gumma, which may affect skin, bones, muscles and various viscera with its subsequent destruction.

Diagnosis

Diagnosis is based on dark field microscopy, non - specific serological test and specific serological test. Dark field microscopic examination reveals the presence of Treponema pallidum in the exudates of the chancre in primary and secondary syphilis. Non- specific serological test like reactive venereal disease research laboratory (VDRL) test indicates the past or present infection with Treponema pallidum and specific serological test like Treponema pallidum hemagglutination assay (TPHA), Treponema pallidum immobilization test (TPI), fluorescent treponemal antibody absorption test (FTA-ABS) usually become positive 3 to 5 weeks after infection.

Differential diagnosis

Differential diagnoses for syphilis encompass Behçet's syndrome, chancroid, contact or atopic dermatitis, erythema multiforme, genital herpes, granuloma inguinale, and lymphogranuloma venereum.

GONORRHOEA

Gonorrhoea is an acute bacterial infection caused by gram-negative diplococcic Neisseria gonorrhoeae and is almost transmitted through sexual contact (vaginal, oral or anal sex). It may involve columnar epithelium of lower genital tract, rectum, pharynx and eyes. Upto 30% of male and female patients with gonorrhoea may also get infected with Chlamydia trachomatis.

Incubation period – usually varies from 2-10 days

Clinical features:

Anterior urethritis is the common feature in the male patient along with purulent urethral discharge, dysuria and erythema of the urethral meatus may occur. Whereas in female patient, the endo-cervical canal is the prime site of infection and urethra may get involved that leads to purulent vaginal discharges, dysuria, frequent urination with burning sensation. Proctitis is common in men who are homosexual and in women due to rectum infected with vaginal secretions.

Complications

In males, the complications include prostatitis, epididymitis, vesiculitis, cystitis and urethral stricture. In females, the complications include vulvitis, bartholinitis, cervicitis, salpingitis and pelvic inflammatory disease.

Diagnosis

Diagnosis is based on gram's stain or culture. Gram stained smear from urethral discharge show gram-negative Neisseria gonorrhoeae. Culture for Neisseria gonorrhoeae on Thayer martin agar remains the gold standard media.

Differential diagnosis

Differential diagnoses for gonorrhea include Chlamydia trachomatis, Trichomonas vaginalis, Treponema pallidum, Mycoplasma genitalium, and herpes simplex virus. Additionally, other conditions such as nongonococcal urethritis, vaginitis, cervicitis, urinary tract infections, prostatitis, and orchitis should also be considered.

MCQ's

1. Which of the following is a bacterial sexually transmitted disease

a) Herpes simplex b) HIV
c) Chlamydia d) Genital warts

2. Which pathogen causes Trichomoniasis

a) Bacteria b) Virus
c) Fungus d) Parasite

3. What is the causative agent of syphilis
   a) Neisseria gonorrhoeae b) Treponema pallidum
   c) Chlamydia trachomatis d) Haemophilus ducreyi

4. What is the typical incubation period for syphilis
   a) 1-2 days b) 3 weeks
   c) 6 months d) 1 year

5. Which stage of syphilis is characterized by a painless papule called a primary chancre
   a) Primary syphilis b) Secondary syphilis
   c) Latent syphilis d) Tertiary syphilis

6. What is a common feature of secondary syphilis
   a) Single painless ulcer b) Diffuse mucocutaneous rash
   c) Chronic granulomatous lesions d) None of the above

7. Which phase of syphilis is infectious and characterized by recurrent secondary infection
   a) Early latent phase b) Late latent phase
   c) Tertiary phase d) Primary phase

8. What is a characteristic lesion of tertiary syphilis
   a) Primary chancre b) Condyloma lata
   c) Gumma d) Maculopapular rash

9. Which non-specific serological test indicates past or present infection with Treponema pallidum
   a) TPHA b) TPI
   c) FTA-ABS d) VDRL

10. What is the causative agent of gonorrhoea
    a) Chlamydia trachomatis b) Treponema pallidum
    c) Neisseria gonorrhoeae d) Haemophilus ducreyi

11. What is the typical incubation period for gonorrhoea
    a) 1-2 days b) 2-10 days
    c) 1-2 weeks d) 1 month

12. What is the prime site of infection in female patients with gonorrhoea
    a) Urethra b) Endo-cervical canal
    c) Rectum d) Pharynx

13. Which complication is common in males with gonorrhoea
    a) Urethral stricture b) Prostatitis
    c) Cystitis d) All of the above

14. Which symptom is common in female patients with gonorrhoea
    a) Testicular pain b) Purulent vaginal discharge
    c) Skin rash d) Severe headache

15. What is a common site of proctitis in men with gonorrhoea
    a) Pharynx b) Eyes
    c) Rectum d) Skin

Answer Keys

1	c	2	d	3	b	4	b	5	a	6	b	7	a	8	c
9	d	10	c	11	b	12	b	13	d	14	b	15	c		

◆

TOPIC 56

Kṛmiroga

Introduction

Kṛmiroga means the disease which arises due to the presence of Kṛmi i.e. microbes and parasites in broad sense. It is Kapha predominant Tridoṣaja Vyādhi. Ācārya Caraka explained Kṛmi roga in Vimāna sthāna while describing the Vyādhitarūpiya (specific features of the patient). Ācārya Suśruta dedicated a separate chapter for Kṛmiroga in Uttara tantra, whereas Vāgbhaṭa explained Kṛmiroga along with Kuṣṭha and Śvitrā due to the similarity in their causative factors.

Nirūkti and Paribhāṣā

The word Kṛmi is derived from the dhātu 'Kṛñ Hiṃsāyām' which means either to produce fatal effects or to destroy.

- क्रामतीति क्रमु पादविक्षेपे । (SKD)

That which moves with the legs is termed as Kṛmi.

- भ्रमेः सम्प्रसारणञ्च इति । (SKD)

That which moves or roams is called Kṛmi.

- लुम्पन्ति कीटकृमयः परितस्तथैते । (Vācaspatyam)

That which is capable to break or injure the surroundings is called as Kṛmi or Kīṭa.

- क्रिमिन् क्रव्ये मेद्यति क्रामते वा स्यात् सरणः कर्मणः कामते वा ॥ (Yāskācārya Niruktam. 6/3/13)

Those which thrive on raw flesh or fat and perchance perform some movements are called Kṛmi.

- कृमिजनितो रोगः कृमिरोग । (Āḍ.Śā.P.7/14-18)

The disease which arises due to presence of Kṛmi is called Kṛmiroga.

Paryāya

Kīṭa and Nīlāṅga are the synonyms of Kṛmi

Sāmānya Nidāna and Saṃprāpti

- अजीर्णाध्यशनासात्म्यविरुद्धमलिनाशनैः । अव्यायामदिवास्वप्नगुर्वतिस्निग्धशीतलैः ॥
  माषपिष्टान्नविदलबिसशालूकसेरुकैः । पर्णशाकसुराशुक्तदधिक्षीरगुडेक्षुभिः ॥
  पललानूपपिशितपिण्याकपृथुकादिभिः । स्वाद्वम्लद्रवपानैश्च श्लेष्मा पित्तं च कुप्यति ॥
  कृमीन् बहुविधाकारान् करोति विविधाश्रयान् । (Su.U.54/3-6)

Ajīrṇa (indigestion), Adhyaśana (eating before the previous meal has been digested), Asātmyaviruddhamalināśanaiḥ (unwholesome, incompatible and unhygienic food substances), Avyāyāma (absence of physical exercise), Divāsvapna (sleeping during day time), Gurvatisnigdhaśītalaiḥ (excessive intake of heavy, unctuous and cold food substances), Māṣa (black gram), Piṣṭānna (cake and pastries), Vidala (legumes), Bisaśālūka (stalk and roots of lotus), Kaserukaiḥ, Parṇāśāka (leafy vegetables), Surā (wine), Saktu (a kind of fermented rice-gruel), Dadhi (curd), Kṣīra (milk), Guḍa (jaggery), Ikṣu (sugar-cane), Palalānūpapiṣita (minced meat of marshy animals), Pinyāka (oil-cake), Pṛthukādibhiḥ (a kind of parched flattened rice) etc., and Svādvamladravapānai (sweet and sour liquid drinks) leads to aggravation of Kapha and Pitta doṣas resulting in production of Kṛmis of various types and shapes in the different parts of the body.

Saṃprāpti Ghaṭaka of Kṛmi:

Doṣa	Kapha Pradhāna Tridoṣa
Dūṣya	Rasa, Rakta and Māṃsa
Agni	Jaṭharāgni māndhya
Āma	Agni māndhya janya Āma
Srotas	Anna, Rasa Rakta and Purīṣa vaha
Srotoduṣṭi	Saṅga and Vimārga gamana
Sañcāra sthāna	Sarvatara
Udbhava sthāna	Āmapakvāśaya
Vyakta sthāna	Guda, Tvaca and Mukha
Adhiṣṭhāna	Āmapakvāśaya and Dhamanī
Rogamārga	Bahya and Abhyantara

Sāmānya Lakṣaṇās

- ज्वरो विवर्णता शूलं हृद्रोगः सदनं भ्रमः ॥ भक्तद्वेषोऽतिसारश्च सञ्जातकृमिलक्षणम् । (Su.U.54/18-19)
- ज्वरो हृद्रोगशूलं वा वमिहृत्क्लेदनं भ्रमः। सुचिबन्धो विवर्णत्वमतीसारः सफेनिलः ॥
  गर्जनं जठरे चैव मन्दग्नित्वं च जायते । पिपासा पीतता नेत्रे किञ्चुकैः पीडितस्य च ॥ (Hā.Sa.Tṛ.5/12-13)

The clinical features of Kṛmi (worm infestations) are Jvara (fever), Vivarṇatā (discoloration), Śūla (colic), Hṛdroga (cardiac disorders), Sadana (malaise), Bhrama (giddiness), Bhaktadveṣā (aversion to food) and Atisāra (diarrhoea).

Ācārya Hārīta added the following features Vami (vomiting), Hṛtkleda, Rucibandha (anorexia), Atīsāraḥ saphenilaḥ (frothy diarrhoea), Jaṭhara garjana (gurgling sound in the abdomen), Mandāgni (suppressed digestive fire), Pipāsā (thirst) and Netrapītatā (yellowish colour of eyes).

Kṛmi Bheda

- अथास्मै प्रोवाच भगवानात्रेयः- इह खल्वग्निवेश! विंशतिविधाः क्रिमयः पूर्वमुद्दिष्टा नानाविधेन प्रविभागेनान्यत्र सहजेभ्यः; ते पुनः प्रकृतिभिर्विभज्यमानाश्चतुर्विधा भवन्ति; तद्यथा- पुरीषजाः, श्लेष्मजाः, शोणितजा, मलजाश्चेति॥ (Ca.Vi.7/9)

Ācārya Caraka described twenty types of Kṛmi and again he classified into two broad groups namely Sahaja Kṛmi and Vaikārikā Kṛmi. Sahaja or Avaikārikā means Sahajebhya vanena śarīrasahajāstva - which lives in human body right from birth and are not harmful to the body i.e. non- pathogenic. Vaikārikā means that which is opposite to the Sahaja Kṛmi, because they are harmful to the body i.e. pathogenic in nature and it is further classified into four types Purīṣajā, Śleṣmajā, Śoṇitajā and Malajā (Bāhyamalajā) based on their sources.

- कृमयस्तु द्विधा प्रोक्ता बाह्याभ्यन्तरभेदतः। बहिर्मलकफासृग्विड्जन्मभेदाच्चतुर्विधाः।
  नामतो विंशतिविधाः..। (A.H.Ni.14/42-43)

Ācārya Vāgbhaṭa classified Kṛmi into two types based on their location, i.e. Bāhya (external) and Abhyantara (internal). Again he classified into four types based on their source of origin, Bahirmala (external excreta like sweat etc.), Kapha, Asṛk (blood) and Viḍ (faeces). Further classified in to twenty types by their names.

- आमपक्वाशये तेषां कफविड्जन्मनां पुनः । धमन्यां रक्तजानां च प्रसवः प्रायशः स्मृतः ॥
  विंशतेः कृमिजातीनां त्रिविधः सम्भवः स्मृतः । पुरीषकफरक्तानि, तासां वक्ष्यामि विस्तरम् ॥ (Su.U.54/6-7)

Ācārya Suśruta classified Kṛmi into twenty types and based on their origin it is classified three types i.e. Kaphaja Kṛmi originate in Āmaśaya (stomach), Viḍ or Purīṣaja Kṛmi originate in Pakvāśaya (intestines) and Raktaja Kṛmi originate in Dhamanī (blood vessels). These three sources, twenty types of Kṛmis are originated. Further while describing the ...osis, he classified into two types based on the visibility i.e. Dṛśya (visible – Kaphaja and ...ja Kṛmi) and Adṛśya (invisible – Raktaja Kṛmi).

Ācārya Hārīta classified Kṛmi in to thirteen types i.e. seven types of Bahya Kṛmi and six types of Abhyantara Kṛmi; whereas Ācārya Śārṅgadhara described twenty two types by adding two types of Kṛmis i.e. Snāyuka Kṛmi and Vraṇa Kṛmi.

Malaja Kṛmi or Bāhya Kṛmi

- तत्र मलो बाह्यश्चाभ्यन्तरश्च। तत्र बाह्यमलजातान् मलजान् सञ्चक्ष्महे । तेषां समुत्थानं- मृजावर्जनं; स्थानं- केशश्मश्रुलोमपक्ष्मवासांसि; संस्थानम्- अणवस्तिलाकृतयो बहुपादाश्च; वर्णः- कृष्णः, शुक्लश्च; नामानि- यूकाः, पिपीलिकाश्च; प्रभावः- कण्डूजननं, कोठपिडकाभिनिर्वर्तनं च; चिकित्सितं तु खल्वेषामपकर्षणं, मलोपघातः, मलकराणां च भावानामनुपसेवनमिति॥ (Ca.Vi.7/10)
- बाह्यास्तत्रामृजोद्भवाः॥ तिलप्रमाणसंस्थानवर्णाः केशाम्बराश्रयाः। बहुपादाश्च सूक्ष्माश्च यूका लिक्षाश्च नामतः॥ द्विधा ते कोठपिटिकाकण्डूगण्डान् प्रकुर्वते। (A.H.Ni.14/45)

Mala (excreta) are both Bāhya (external) and Abhyantara (internal). Here the word Malaja Kṛmi referring the Bāhya Kṛmi (external parasites) only.

- **Samutthāna (causative factor) :** Mṛjā varjanaṃ (avoidance of cleanliness of the body). Vāgbhaṭa described Amṛjodbhavā (originates from impurities or dirt).
- **Sthāna (location) :** Keśa (hair), Śmaśru (beards), Loma (body hair), Pakṣma (eyelashes) and Vāsāṃsi (clothes or garments). Vāgbhaṭa described Keśāmbarāśrayāḥ (resides in hairs and clothes)
- **Saṃsthāna (shape) :** Aṇavaḥ (minute), Tilākṛtayaḥ (sesame seed-shaped) and Bahupāda (many- legged / multiped)
- **Varṇa (colour) :** Kṛṣṇa (black) and Śukla (white). Vāgbhaṭa mentioned Tilāpramāṇa (sesame seed-color)
- **Nāma (name) :**

Bāhya Kṛmi	Caraka	Suśruta	Vāgbhaṭa
Yūkā	+	–	+
Pipīlikā	+	–	–
Likṣā	–	–	+

- **Prabhāva (effect) :** Kaṇḍū (itching), Koṭha (urticarial patches) and Piḍakā (pimples). Vāgbhaṭa added Piṭikā (eruptions) and Gaṇḍā (tumour))

Śoṇitaja Kṛmi

- शोणितजानां तु खलु कुष्ठैः समानं समुत्थानं; स्थानं- रक्तवाहिन्यो धमन्यः; संस्थानम्- अणवो वृत्ताश्चापादाश्च, सूक्ष्मत्वाच्चैके भवन्त्यदृश्याः; वर्णः- ताम्रः; नामानि- केशादा, लोमादा, लोमद्वीपाः, सौरसा, औडुम्बरा, जन्तुमातरश्चेति; प्रभावः- केशश्मश्रुनखलोमपक्ष्मापध्वंसः, व्रणगतानां च हर्षकण्डूतोदसंसर्पणानि, अतिवृद्धानां च त्वक्सिरास्नायुमांसतरुणास्थिभक्षणमिति; चिकित्सितमप्येषां कुष्ठैः समानं, तदुत्तरकालमुपदेक्ष्यामः॥ (Ca.Vi.7/11)

- ⊙ रक्तवाहिसिरोत्थाना रक्तजा जन्तवोऽणवः। अपादा वृत्तताम्राश्च सौक्ष्म्यात्केचिददर्शनाः॥
  केशादा लोमविध्वंसा लोमद्वीपा उदुम्बराः। षट् ते कुष्ठैककर्माणः सहसौरसमातरः॥ (A.H.Ni.14/51-52)
- ⊙ कुष्ठैकहेतवोऽन्तर्जाः ..... । (A.H.Ni.14/45)
- ⊙ विरुद्धाजीर्णशाकाद्यैः शोणितोत्था भवन्ति हि । (Su.U.54/18)
- ⊙ केशरोमनखादाश्च दन्तादाः किक्किशास्तथा । कुष्ठजाः सपरीसर्पा ज्ञेयाः शोणितसम्भवाः ॥
  ते सरक्ताश्च कृष्णाश्च स्निग्धाश्च पृथवस्तथा । रक्ताधिष्ठानजान् प्रायो विकारान् जनयन्ति ते ॥ (Su.U.54/15-16)
  - ◈ **Samutthāna (causative factor) :** Kuṣṭhaiḥ Samānaṃ (similar to those of Kuṣṭha) Suśruta described Viruddhājīrṇaśākādyaiḥ (incompatible diet and uncooked leafy vegetables)
  - ◈ **Sthāna (location) :** Raktavāhinyo dhamanyaḥ / Raktavāhisirotthānā (blood carrying vessels or arteries)
  - ◈ **Saṃsthāna (shape) :** Aṇavaḥ (minute), Vṛta (round), Apāda (devoid of legs), Sūkṣma / Saukṣmyāt (very minute), Adṛṣya / Adarśana (invisible). Suśruta added Snigdha (unctuous) and Pṛthu (wide /large)
  - ◈ **Varṇa (colour) :** Tāmra (coppery color)) Suśruta mentioned Sarkta (tinged with blood) and Kṛṣṇa (black)

Nāma (name)

Śoṇitaja Kṛmi	Caraka	Suśruta	Vāgbhaṭa
Keśada	+	+	+
Lomāda / Romāda / Romavidhvāṃśa	+	+	+
Lomadvīpa	+	–	+
Saurasa / Aurasa	+	–	+
Auḍumbara / Udumbara	+	–	+
Jantumātṛ / Mātrata	+	–	+
Nakhada	–	+	–
Dantāda	–	+	–
Kikkiśa	–	+	–
Kuṣṭhaja	–	+	–
Parīsarpa	–	+	–

- ⊙ **Prabhāva (effect)** : Keśaśmaśrunakhalomapakṣmāpadhvaṃsa (falling of hair, moustache, nails, body hair and eyelashes), Vraṇagatānāṃ ca

itching, pricking pain and creeping sensation) and Ativṛddhānāṃ ca tvaksirāsnāyumāṃsataruṇāsthibhakṣaṇamiti (when excessively increased these eat away the skin, veins, ligaments, muscles and cartilages)

Śleṣmaja Kṛmi

- ⊙ श्लेष्मजा:क्षीरगुडतिलमत्स्यानूपमांसपिष्टान्नपरमान्नकुसुम्भस्नेहाजीर्णपूतिक्लिन्नसङ्कीर्णविरुद्धासात्म्य भोजनसमुत्थानाः; तेषामामाशयः स्थानं, ते प्रवर्धमानास्तूर्ध्वमधो वा विसर्पन्त्युभयतो वा; संस्थानवर्णविशेषास्तु- श्वेताः पृथुब्रध्नसंस्थानाः केचित्, केचिद्वृत्तपरिणाहा गण्डूपदाकृतयः श्वेतास्ताम्रावभासाश्च, केचिदणवो दीर्घास्तन्त्वाकृतयः श्वेताः; तेषां त्रिविधानां श्लेष्मनिमित्तानां क्रिमीणां नामानि- अन्त्रादाः, उदरादाः, हृदयचराः , चुरवः, दर्भपुष्पाः, सौगन्धिकाः, महागुदाश्चेति; प्रभावो- हृल्लासः, आस्यसंस्रवणम्, अरोचकाविपाकौ, ज्वरः, मूर्च्छा, जृम्भा, क्षवथुः, आनाहः, अङ्गमर्दः, छर्दिः कार्श्यं, पारुष्यं, चेति॥ (Ca.Vi.7/12)
- ⊙ कफादामाशये जाता वृद्धाः सर्पन्ति सर्वतः। पृथुब्रध्ननिभाः केचित् केचिद्गण्डूपदोपमाः॥
  रूढधान्याङ्कुराकारास्तनुदीर्घास्तथाऽणवः। श्वेतास्ताम्रावभासाश्च नामतः सप्तधा तु ते॥
  अन्त्रादा उदरावेष्टा हृदयादा महाकुहाः। कुरवो दर्भकुसुमाः सुगन्धास्ते च कुर्वते॥
  हृल्लासमास्यस्रवणमविपाकमरोचकम्। मूर्च्छाच्छर्दिज्वरानाहकार्श्यक्षवथुपीनसान्॥ (A.H.Ni.14/47-50)
- ⊙ .....श्लेष्मजास्तेषु चाधिकम्॥ मधुरान्नगुडक्षीरदधिसक्तुनवौदनैः। (A.H.Ni.14/45-46)
- ⊙ मांसमाषगुडक्षीरदधितैलैः कफोद्भवाः ॥ (Su.U.54/17)
- ⊙ दर्भपुष्पा महापुष्पाः प्रलूनाश्चिपिटास्तथा । पिपीलिका दारुणाश्च कफकोपसमुद्भवाः ॥
  रोमशा रोममूर्धानः सपुच्छाः श्यावमण्डलाः । रूढधान्याङ्कुराकाराः शुक्लास्ते तनवस्तथा ॥
  मज्जादा नेत्रलेढारस्तालुश्रोत्रभुजस्तथा । शिरोहृद्रोगवमथुप्रतिश्यायकराश्च ते ॥ (Su.U.54/12-14)
  - ◈ **Samutthāna (causative factor) :** indulging in Kṣīra (milk), Guḍa (jaggery), Tila (sesame), Matsya (fish), Ānūpa Māṃsa (meat of marshy land animals), Piṣṭānna (food preparations made with ground pulses), Paramānna (Pāyasa - rice cooked with milk and sweetened), Kusumbha sneha (oil of kusumbha), Ajīrṇa (uncooked), Pūti (putrefied), Klinna (stale), Saṅkīrṇa (food mixed with abhorrent articles such as faeces etc.), Viruddha (contrary food), Asātmya bhojana (unwholesome food).
  - ◈ **Sthāna (location) :** Āmāśaya (stomach), when grown it moves Ūrdhva (upwards), Adha (downwards) or Ubhaya (both the ways)
  - ◈ **Saṃsthāna (shape) and Varṇa (colour) :** Some are Pṛthu Bradhna (wide /large like Carmalatā (leather strip)) and Śveta (white); some are Vṛtta (round) like Gaṇḍupadakṛta (tape worm) having Śvetatāmra varṇa (white or coppery colour); some are Aṇava (small), Dīrghāstantvākṛta (long and thread like) and Śveta (white) in colour. Vāgbhaṭa and Suśruta added Rūḍhadhānyāṅkurākāra (sprouts of germinating grains)

Nāma (name)

Kaphaja Kṛmi	Caraka	Suśruta	Vāgbhaṭa
Antrāda	+	–	+
Udarāda / Udarāveṣṭa	+	–	+
Hṛdayacara / Hṛdayāda	+	–	+
Curu	+	–	–
Darbhapuṣpa / Darbhakusuma	+	+	+
Saugandhika / Sugandha	+	–	+
Mahāguda / Mahākuha	+	–	+
Mahāpuṣpa	–	+	–
Pralūna	–	+	–
Cipiṭa	–	+	–
Pipīlikā	–	+	–
Dāruṇa	–	+	–
Kurava	+	–	+

- **Prabhāva (effect) :** Hṛllāsa (nausea), Āsyasravaṇa (salivation), Avipāka (indigestion), Arocaka (anorexia), Jṛmbhā (yawning), Mūrcchā (fainting), Chardi (vomiting), Jvara (fever), Ānāha (constipation), Aṅgamarda (body ache), Kārśya (emaciation), Kṣavathu (sneezing), and Pāruṣya (roughness). Vāgbhaṭa added Pīnasa (nasal catarrh) and Suśruta added Śiroroga (diseases of head), Hṛdroga (diseases of heart), and Pratiśyāya (coryza)

Purīṣaja Kṛmi

- पुरीषजास्तुल्यसमुत्थानाः श्लेष्मजैः; तेषां स्थानं पक्वाशयः, ते प्रवर्धमानास्त्वधो विसर्पन्ति, यस्य पुनरामाशयाभिमुखाः स्युर्यदन्तरंतदन्तरं तस्योद्गारनिःश्वासाः पुरीषगन्धिनः स्युः; संस्थानवर्णविशेषास्तु- सूक्ष्मवृत्तपरीणाहाः श्वेता दीर्घा ऊर्णांशुसङ्काशाः केचित्, केचित् पुनः स्थूलवृत्तपरीणाहाः श्यावनीलहरितपीताः; तेषां नामानि ककेरुकाः, मकेरुकाः, लेलिहाः; सशूलकाः, सौसुरादाश्चेति; प्रभावः- पुरीषभेदः, कार्श्यं, पारुष्यं, लोमहर्षाभिनिर्वर्तनं च, त एव चास्य गुदमुखं परितुदन्तः कण्डूं चोपजनयन्तो गुदमुखं पर्यासते, त एव जातहर्षा गुदनिष्क्रमणमतिवेलं कुर्वन्ति; इत्येष श्लेष्मजानां पुरीषजानां च क्रिमीणां समुत्थानादिविशेषः॥ (Ca.Vi.7/13)
- पक्वाशये पुरीषोत्था जायन्तेऽधोविसर्पिणः। वृद्धाः सन्तो भवेयुश्च ते यदाऽऽमाशयोन्मुखाः।
  तदाऽस्योद्गारनिःश्वासा विड्गन्धानुविधायिनः। पृथुवृत्ततनुस्थूलाः श्यावपीतसितासिताः।
  ते पञ्च नाम्ना कृमयः ककेरुकमकेरुकाः। सौसुरादः सुलूनाख्या लेलिहा जनयन्ति च।
  विड्भेदशूलविष्टम्भकार्श्यपारुष्यपाण्डुताः। रोमहर्षाग्निसदनगुदकण्डूर्विनिर्गमात्। (A.H.Ni.14/53-56)
- माषपिष्टान्नविदलपर्णशाकैः पुरीषजाः । (Su.U.54/17)
- शकृज्जा बहुविड्धान्यपर्णशाकोलकादिभिः॥ (A.H.Ni.14/46)

- अजवा विजवाः किप्याश्चिप्या गण्डूपदास्तथा । चूरवो द्विमुखाश्चैव ज्ञेयाः सप्त पुरीषजाः ॥
  श्वेताः सूक्ष्मास्तुदन्त्येते गुदं प्रतिसरन्ति च । तेषामेवापरे पुच्छैः पृथवश्च भवन्ति हि ॥
  शूलाग्निमान्द्यपाण्डुत्वविष्टम्भबलसङ्क्षयाः । प्रसेकारुचिहृद्रोगविड्भेदास्तु पुरीषजैः ॥ (Su.U.54/8-10)
- **Samutthāna (causative factor) :** Śleṣmaja Samānaṃ (similar to those of Kaphaja Kṛmī). Suśruta and Vāgbhaṭa described Māṣa (black gram), Piṣṭānna (cake and pastries), Vidala (legumes), Bahuviḍ dhānya (grains which produces more faeces), Parṇaśākā (leafy vegetables) and Okulādi (slightly fried wheat, barley etc.)
- **Sthāna (location) :** Pakvāśaya (intestines) which moves downwards usually, when increased in number, they moves upwards to Āmāśaya (stomach) and produces Viḍ gandha (smell of faeces) in the mouth during Udgāra (eructation) and Niḥśvāsā (expiration)
- **Saṃsthāna (shape) :** Pṛthu (thick), Vṛtta (round), Tanu (thin), Dīrghā (long), Ūrṇāśusaṅkāśā (resembling fiber of wool), and Sthūla (large). Suśruta described Sūkṣmā (small in size), Guda pratisaranti (spreads towards the anus), and some of them have Pṛthava pucchaiḥ (thick tails)
- **Varṇa (colour) :** Śyāva (dark brown), Nīla (blue), Harita (green), Pītā (yellow), Sitāsita (white or black in colour). Suśruta mentioned Svetā (white)

Nāma (name)

Purīṣaja Kṛmi	Caraka	Suśruta	Vāgbhaṭa
Kakerukā	+	–	+
Makerukā	+	–	+
Leliha	+	–	+
Saśūlakā / Sulūna	+	–	+
Sausurādā	+	–	+
Ajavā	–	+	–
Vijavā	–	+	–
Kipyā	–	+	–
Cipyā	–	+	–
Gaṇḍūpada	–	+	–
Cūrava	–	+	–
Dvimukhā	–	+	–

- **Prabhāva (effect) :** Purīṣabheda (diarrhoea), Kārśya (emaciation), Pāruṣya (roughness of the skin), Lomaharṣā (horripilation), Gudamukhaṃ paritudantaḥ kaṇḍūṃ copajanayanto gudamukhaṃ paryāsate (when they reach anal region produces piercing and itching sensation in the anal orifice) and Jātaharṣā gudaniṣkramaṇātivelaṃ (exhilarated when these comes out of anus). Vāgbhaṭa added

Śūla (colic), Viṣṭambha (constipation), Pāṇḍutāḥ (anaemia), Agnisadana (weakness of digestive fire) and Gudakaṇḍuvinirgamāt (itching sensation of the anus while coming out).

Suśruta added Balasaṃkṣayā (loss of strength), Praseka (salivation), Aruci (anorexia), and Hṛdroga (cardiac disorders). Gaṇḍūpada Kṛmi is Raktā (red in colour) and Dīrghā (long in their shape); and it produces Gudakaṇḍunipātinaḥ (itching sensation of the anus while coming out), Śūla (colic), Ātopa (distension of abdomen), Śakṛtbheda (diarrhoea) and Paktināśa (destroys the power of digestion)

Sādhyāsādhyata

◉ दृश्यास्त्रयोदशाद्यास्तु कृमीणां परिकीर्तिताः ॥ केशादाद्यास्त्वदृश्यास्ते द्वावाद्यौ परिवर्जयेत् । (Su.U.54/19-20)

First thirteen types, i.e. Purīṣaja and Kaphaja Kṛmīs are Dṛśya (visible). Keśādā, etc., Śoṇitaja Kṛmīs are Adṛśya (invisible). Keśādā and Romāda Kṛmīs should be discarded for treatment as it is incurable.

In Contemporary science

Kṛmi roga can be correlated with parasitic disorders in contemporary science. (Please refer topic 57).

MCQ's

1. Kīta and Nīlāṅga are the synonym of

a) Kṛmiroga b) Śvitra
c) Kuṣṭha d) All of the above

2. According to Śārṅgadhara, Kṛmi is of .... types

a) 2 b) 13
c) 20 d) 22

3. According to Caraka, Kṛmi is of .... types

a) 2 b) 13
c) 20 d) 22

4. According to Vāgbhaṭa, Kṛmi is of .... types based on their source of origin

a) 2 b) 4
c) 20 d) 22

5. Sthāna of Kaphaja Kṛmi

a) Āmāśaya b) Pakvāśaya
c) Dhamanī d) Keśasmaśru

6. Adṛśya Kṛmis are

a) Bāhya Kṛmi b) Śoṇitaja Kṛmi
c) Śleṣmaja Kṛmi d) Puriṣaja Kṛmi

7. Yūka and Pipīlikā are the types of

a) Bāhya Kṛmi b) Śoṇitaja Kṛmi
c) Śleṣmaja Kṛmi d) Puriṣaja Kṛmi

8. According to Caraka, Śoṇitaja Kṛmi is of ....

a) 2 b) 4
c) 6 d) 8

9. Which type of Kṛmi resembles रूढधान्याङ्कुराकारा

a) Bāhya Kṛmi b) Śoṇitaja Kṛmi
c) Śleṣmaja Kṛmi d) Puriṣaja Kṛmi

10. गुदमुखं परितुदन्तः कण्डूं is seen in

a) Bāhya Kṛmi b) Śoṇitaja Kṛmi
c) Śleṣmaja Kṛmi d) Puriṣaja Kṛmi

Answer Keys

1	a	2	d	3	c	4	b	5	a	6	b	7	a	8	c	9	c	10	d

TOPIC 57

Clinical Presentation of Common Parasitic Disorders

Introduction

Parasitic disorders, or Parasitosis, are a group of infectious diseases caused by parasitic organisms that depend on a host organism to survive and reproduce. These organisms extract nutrients from the host while causing damage to their tissues and organs. They can be broadly categorized into three main types: protozoa, helminths, and ectoparasites. Protozoa and helminths, known as endoparasites, typically live inside the host's body.

- **Protozoa** are single-celled organisms that can multiply within their host. They are responsible for diseases such as, malaria and amoebiasis.
- **Helminths** are larger, multicellular organisms, often visible to the naked eye. They include roundworms, tapeworms, and flukes.
- **Ectoparasites** are indeed parasites that live on the external surface of their host, such as lice, fleas, and mites.

They are the cause of many diseases and are primarily transmitted to their hosts through the consumption of contaminated food or water, or through the bite of an arthropod, such as a fly or tick, which serves as an intermediate host and a vector.

This chapter deals explicitly with helminthiasis, an infection caused by helminths (worms) among the three parasitic organisms.

Helminthiasis

Helminthiasis refers to diseases caused by helminths (worms). It is estimated that over 1.5 billion people worldwide are affected by helminths and India contributes 25% of the global cases. Helminths affecting man can be divided into three groups nematodes (roundworms), trematodes (flukes) and cestodes (tapeworms).

- **Nematodes (roundworms):**
  - **Intestinal human nematodes** such as Enterobius vermicularis, Ascaris lumbricoides, Trichuris trichiura, Necator americanus, Ankylostoma duodenale, etc.
  - **Tissue-dwelling human nematodes** such as Wuchereria bancrofti, Brugia malayi, Loa loa, Onchocerca volvulus, etc.
- **Trematodes (flukes):**
  - **Blood flukes** such as Trichobilharzia regenti, Schistosoma guineensis, Schistosoma haematobium, etc.
  - **Lung flukes** such as Paragonimus westermani,
  - **Liver flukes** such as Fasciola hepatica, Clonorchis sinensis, Dicrocoelium dendriticum, Dicrocoelium hospes, Fasciola gigantica, etc.
  - **Intestinal flukes** such as Fasciolopsis buski, Metagonimus miyatai, Metagonimus takahashii, etc.
- **Cestodes (tapeworms):**
  - **Intestinal Cestodes** such as Taenia solium, Taenia saginata
  - **Tissue Cestodes** such as Echinococcus granulosus, Echinococcus multilocularis, Echinococcus vogeli, etc.

Infections can occur through ingestion of contaminated food or water, contact with infected soil, or transmission by insect vectors

HOOK WORM (ANCYLOSTOMIASIS)

Hookworm infections are indeed common in tropical and subtropical regions with warm, moist climates. It is caused by two species of hookworms namely Ancylostoma duodenale and Necator americanus, both of which affect the small intestine of human and extracts blood (0.2ml/day) leading to anaemia. While Ancylostoma ceylanicum, Ancylostoma caninum, and Ancylostoma braziliens are less commonly involved in human infections.

The estimated global prevalence of hookworm infection ranges from 576 to 740 million people, with the highest prevalence observed in sub-Saharan Africa, Asia, Latin America, and the Caribbean. Recent studies indicate that the prevalence of hookworm infections in India is approximately 10%.

Morphology: The adult Ancylostoma duodenale worm is 8-13 mm long and has a thin, thread-like appearance. Its buccal cavity has four pointed, hook-like teeth. The smaller Necator americanus worm has a buccal cavity with two chitinous cutting plates. A fertilized female A. duodenale worm can lay up to 20,000 eggs a day, each measuring 40-60 μ and embryonated when passed. In comparison, Necator americanus lays fewer eggs than A. duodenale, but the eggs are larger, measuring 60-70 μ

Life cycle: The life cycle of the hookworm starts with the infective larvae passing from the contaminated soil, which penetrates the human skin, typically through bare feet. Then, the larvae enter the bloodstream and travel to the lungs. From there, the larvae move up to the bronchi and are eventually swallowed. They reach the duodenum and jejunum, where they mature into adult worms. Their eggs are passed out in the faeces and develop into the filariform infective larval stage in the soil.

Risk factors: The risk factors for this condition include low socioeconomic background, exposure to contaminated soil, walking barefoot, inadequate sanitation, and substandard personal hygiene. Among those at the highest risk are children and pregnant women. The transmission of the affliction is influenced by a combination of factors including warm and moist climates, contaminated water sources, and poor sanitation practices.

Clinical features: Exposed skin may become pruritic and damp, potentially leading to local abscess formation if secondary infection occurs. Larval migration through the lungs can elicit allergic symptoms, general discomfort, dyspnoea, cough, and haemoptysis (Loeffler's syndrome). Intestinal infestation may result in blood loss, contributing to iron deficiency anaemia, which may be exacerbated by concurrent malnutrition and the manifestation of abdominal symptoms such as pain, burning sensation, and distension.

Diagnosis: The diagnosis is based on the microscopic examination of stool. This examination can be done by using the Kato-Katz cellophane thick smear technique or by Stoll's method to count the number of eggs, especially in cases of heavy infections. Sputum examination may reveal occult blood, eosinophils, and occasionally migrating larvae. A skin test antigen derived from Necator americanus is sometimes utilized for screening populations.

ROUND WORM (ASCARIASIS)

Ascariasis, the most common intestinal worm infection in the world, is caused by the largest intestinal nematode, Ascaris lumbricoides. It predominantly impacts children and adults in tropical and subtropical regions characterised by inadequate sanitation, poor personal hygiene, and the widespread use of human faeces as organic fertiliser.

The estimated global prevalence of Ascariasis stands at approximately 11.01%, afflicting an estimated 732 million individuals. Within India, the prevalence of Ascariasis varies significantly, ranging from 0.6% to 91% across different regions. Notably, India accounts for nearly a quarter of global cases, with approximately 220.6 million children necessitating preventive chemotherapy.

Morphology: The length of the worm ranges from 20 to 35 cm, with the females being larger than the males. Each day, the females lay several thousand eggs, which are elliptical in shape and measure 30-40 μ x 50-60 μ. These eggs possess an outer dense mammilated shell and a smooth translucent inner shell. They are excreted in faeces and become embryonated and infectious in the soil within 2 to 3 weeks. Under optimal conditions, these eggs can remain viable for numerous years.

Life cycle: The life cycle of Ascaris begins when mature eggs are ingested through contaminated food or water. Upon reaching the small intestine, the eggs hatch into larvae, and penetrate the intestinal mucosa and enter the portal circulation, travelling through the liver to the heart and lungs. After lodging in the alveolar capillaries, they breach the alveolar walls and ascend the bronchial tree to the oropharynx. Upon being swallowed, the larvae return to the small bowel, where they undergo development into adult worms. The mature worms then reproduce and release eggs into the stool.

Clinical features: Ascariasis is often asymptomatic, but when symptoms do occur, they can be quite varied depending on the stage of the parasite's life cycle and the organs involved. When Ascaris larvae migrate through the lungs, they may cause fever, non-productive cough, dyspnoea, wheezing and occasionally haemoptysis. As the larvae migrate through the intestine and mature into adult worms, they can cause diffuse abdominal pain, nausea, vomiting, biliary and intestinal obstruction, volvulus, intussusceptions, appendicitis, pancreatitis and passage of worms (from mouth, nose and anus).

Diagnosis: The diagnosis is based on the history of passage of worms either in the stool or even passed out from the mouth and diagnosis is confirmed by demonstrating the characteristic ova in faeces. Complete blood count may show peripheral eosinophilia and leucocytosis especially during the stage of larval migration.

THREAD WORM / PIN WORM (ENTEROBIASIS)

Enterobiasis is a common nematode infection caused by the pinworm or thread worm Enterobius vermicularis and is most commonly found in children. Risk factors for pinworms include poor hygiene, eating after touching contaminated items, living with someone who is identified as egg-positive, and engaging in anal-oral contact with an infected sexual partner.

The World Health Organization (WHO) has reported a global prevalence of Enterobiasis in children ranging from 4% to 28%. In India, the prevalence of Enterobiasis varies by region, but is generally low compared to other parts of the world. Studies suggest that the prevalence in Indian children is 10-15%.

Morphology: The adult female pinworm measures 8 to 13 mm in length and 0.5 mm in thickness, featuring a distinctly pointed posterior end. In comparison, the adult male is significantly smaller, with dimensions of 2 to 5 mm in length and 0.2 mm in thickness, and possesses a curved posterior end. The translucent eggs are 50 to 60μm by 20 to 30μm in size and are characterized by a thick shell flattened on one side. These eggs are typically invisible to the naked eye except in clusters. Notably, the eggs may harbour a developing embryo or a fully formed pinworm larva, which can reach a length of 140–150μm upon maturation.

Life cycle: The life cycle of Enterobius begins with the ingestion of mature eggs, which require approximately 1 to 2 months to mature into adult worms within the small intestine. Subsequently, the gravid female migrates to the anal area mainly at night time and deposit thousands of eggs in the perianal region. This migration causes severe itching, sleeplessness and irritability. This leads to contamination of the fingers and subsequent ingestion of the eggs (autoinfection) and restarting the worm's life cycle. Infrequently, the larvae may re-enter the rectum and the small intestine, reinitiating the life cycle (retro infection).

Clinical features: Enterobiasis is often asymptomatic in most of the cases. However, common symptoms are perianal itching, pruritus and discomfort. Persistent scratching can result in excoriation and eczema in the affected area. Additional manifestations may include frequent urination, vesical irritability, pruritus vulvae, and vaginal discharge in young girls, as well as enuresis and sleep disturbances in children. Notably, instances of appendicitis have revealed pinworm obstruction within the appendiceal lumen, although the parasite's presence may be incidental.

Diagnosis: The diagnosis relies on the identification of eggs, typically achieved through a scotch tape swab taken from the perianal skin. A cellophane tape test involves applying an adhesive tape-like material to the perianal area, followed by a microscopic examination to detect the eggs. Adult worms may sometimes be observable in faecal matter, potentially by the patient.

MCQ's

1. Which of the following is a single-celled organism
   a) Helminths b) Ectoparasites
   c) Protozoa d) Nematodes
2. Which type of parasitic organism includes roundworms, tapeworms, and flukes
   a) Protozoa b) Helminths
   c) Ectoparasites d) Arthropods
3. Which of the following is an example of an intestinal human nematode
   a) Schistosoma haematobium b) Enterobius vermicularis
   c) Fasciola hepatica d) Echinococcus granulosus
4. Which of the following is a blood fluke
   a) Paragonimus westermani b) Fasciola hepatica
   c) Schistosoma haematobium d) Taenia solium

5. How can helminth infections be transmitted
   a) Ingestion of contaminated food or water b) Contact with infected soil
   c) Transmission by insect vectors d) All of the above
6. Which species of hookworm is most commonly involved in human infections
   a) Ancylostoma ceylanicum b) Ancylostoma caninum
   c) Ancylostoma duodenale d) Ancylostoma braziliens
7. Which of the following is a characteristic feature of the Ancylostoma duodenale worm
   a) Two chitinous cutting plates b) Four pointed, hook-like teeth
   c) Buccal cavity with no teeth d) No buccal cavity
8. What is the primary mode of transmission for hookworm larvae to humans
   a) Ingestion of contaminated food b) Penetration through the skin
   c) Inhalation of larvae d) Direct contact with infected animals
9. What is a common clinical feature of hookworm infection
   a) Skin rash b) Iron deficiency anaemia
   c) High fever d) Severe headache
10. Which syndrome is associated with larval migration through the lungs in hookworm infection
    a) Guillain-Barré syndrome b) Loeffler's syndrome
    c) Down syndrome d) Marfan syndrome
11. What is a common symptom of Enterobiasis
    a) Severe headache b) Perianal itching
    c) High fever d) Abdominal pain

Answer Keys

1	c	2	b	3	b	4	c	5	d	6	c
7	b	8	b	9	b	10	b	11	b		

Short Answer Questions

1. Describe Parasitosis
2. Describe Ancylostomiasis
3. Describe Ascariasis
4. Describe Enterobiasis

◆

TOPIC 58

Khālitya and Pālitya

KHĀLITYA

Introduction

Khālitya and Pālitya are Pitta Pradhāna Tridoṣaja Vyādhi, characterised by hair loss or baldness and premature greying of hair, respectively. Ācārya Caraka explained it under Ūrdhvajatrugata roga. Ācārya Vāgbhaṭa explained it under Śirogata rogās. Ācārya Suśruta explained under Kṣudra rogās and mentioned that this disease is not observed in females due to Atyantasukumārāṅga (extremely delicate bodies), Avyāyāmaratā (lack of physical exercises) and Rajoduṣṭaṃ sravanti (menstrual bleeding). Khālitya and Pālitya are generally described as Cāndilatva, likening the smooth, hairless scalp to the moon's surface.

Nirūkti and Paribhāṣa of Khālitya

- ⊙ खलितनिर्लोमशिरस्त्वम् । (In.A.S.Śā.2/51)

That which causes Nirlomaśirastvam (the head being without hair) is called Khālitya.

- ⊙ खलितः शीर्णरोमत्वम्। (In.A.S.Śā.8/11)

That which causes Śīrṇaromatva (shedding of hair) is called Khālitya.

- ⊙ खालित्यं पिञ्जरत्वं च केशानां पतनं तथा॥ (Ca.Sū.5/30)

That which causes Piṅjaratva (greying of hair) and Keśānāṃ patinaṃ (falling of hair) is called Khālitya.

- ⊙ खालित्यं खल्वाटत्वं । (Ḍa.Su.Sū.35/29)

That which causes Khalvāṭatvaṃ (bald-headed) is called Khālitya.

- ⊙ खलतेरपि जन्मैवं शातनं तत्र तु क्रमात्॥ (A.S.U.27/20)
- ⊙ खलतौ, क्रमेण केशानां शातो । (Aru. A.H.U.23/26-27)

Khālitya refers to the Janmaivaṃ śātanaṃ tatra tu kramāt (gradual process of hair falling from its origin).

Synonyms of Khālitya

- ⊙ खालित्यं रुह्येति च तस्य पर्यायकथनम्। तथाच भोजः– "तदिन्द्रलुप्तमित्याहुः खल्लीं रुह्यां च केचन।" इति। (Ma.M.N.55/28-29)

Indralupta, Ruhyā, Rujyā, Khalati, Khallī, Cāca, and Khalita are synonyms for Khālitya

Nirūkti and Paribhāṣa of Pālitya

- ⊙ पलितं केशशुक्लत्वं । (Ḍa.Su.Sū.35/29)

That which causes Keśaśuklatva (whiteness of hair) is called Palita.

- ⊙ पलितं केशशौक्ल्यम्। (In.A.S.Śā.8/11)

That which causes Keśaśauklyam (whiteness of hair) is called Palita.

- ⊙ पलितं श्वेतलोमत्वम् । (In.A.S.Śā.2/51)

That which causes Śvetalomatva (whiteness of hair) is called Palita.

- ⊙ केशानां शुक्लभावेन पलितमित्युच्यते ॥ (Āḍ.Śā.P.7/151-152)

Keśāṇāṃ Śuklabhāva (whiteness of hair) is called Palita.

Nidāna and Saṃprāpti of Khālitya and Pālitya

- ⊙ तस्यौष्ण्यात् पित्तला भवन्त्युष्णासहा, उष्णमुखाः, सुकुमारावदातगात्राः .... क्षिप्रवलीपलितखालित्यदोषाः ॥ (Ca. Vi.8/97)

Ācārya Caraka, while explaining the characters of the Pittaja Prakṛti individual, mentioned that due to Uṣṇa guṇa (hotness) of Pitta, the Khālitya (baldness) appears in the earlier stage of life.

- ⊙ पित्तलैराहारैर्मात्रा सेवितैः खलतिः-खल्वाटः, पिङ्गो वा स्यात्। (Aru.A.H.Śā.1/48)

Excessive intake of Pitta-aggravating foods can indeed lead to conditions like Khalvāṭa (bald-headed) and Piṅga (yellowish or brownish complexion).

- ⊙ लवणनित्या शीघ्रवलीपलितं खालित्यरोगिणं वा । (Ca.Śā.8/21)

Ācārya Caraka, while explaining the Garbhopaghātakara bhāvas (factors injurious to the foetus), mentioned that regular intake of Lavaṇa (saline) substances by pregnant women causes Valī (wrinkling), Palita (greying of hair), and Khālitya (baldness) early in her progeny.

- ⊙ ये ह्यतिलवणसात्म्याः पुरुषास्तेषामपि खालित्यपालित्यानि वलयश्चाकाले भवन्ति॥ (Ca.Vi.1/18)
- ⊙ क्षारः पुनरौष्ण्यतैक्ष्णयलाघवोपपन्नः ..... खालित्यपालित्यभाजो (Ca.Vi.1/17)

Excessive intake of Lavaṇa (salt) and Kṣāra (alkali) leads to Khālitya

- ⊙ धूमातपतुषाराम्वुक्रीडातिस्वप्नजागरैः। उत्स्वेदाधिपुरोवातबाष्पनिग्रहरोदनैः॥ अत्यम्बुमद्यपानेन कृमिभिर्वेगधारणैः। उपधानमृजाभ्यङ्गद्वेषाधःप्रततेक्षणैः॥ असात्म्यगन्धदुष्टामभाष्याद्यैश्च शिरोगताः। जनयन्त्यामयान् दोषाः ......॥ (A.H.U.23/1-3)

Ācārya Vāgbhaṭa explained this disease under Śirogata roga. Hence, the general causative factors of Śirogata roga can be considered as a causative factor of Khālitya and Pālitya. The causative factors are Dhūmātapatuṣāra (exposure to smoke, sunlight and mist), Ambukrīḍāti (excessive indulgence in water sports), Svapnajāgaraiḥ (sleep and waking at night), Utsveda (excessive sweating), Ādhipurovāta (exposure to heavy eastern breeze), Bāṣpanigraha (suppressing tears), Rodanaiḥ (excessive crying), Atyambumadyapāna (excessive intake of water and alcohol), Kṛmibhi (worm infestation), Vegadhāraṇaiḥ (suppressing urges), Upadhānadveṣa (avoiding pillow), Mṛjadveṣa (avoiding cleanliness), Abhyaṅgadveṣa (avoiding anointing the body), Adhaḥ pratatekṣaṇaiḥ (looking downwards for long time), Asātmya gandhaduṣṭāma (exposure to unaccustomed, unhealthy, vitiated or raw smell), and Bhāṣyādyaī (excessive talking). By these above said Nidāna, the doṣa get aggravated and produces the disease Khālitya and Pālitya.

Saṃprāpti Ghaṭaka of Khālitya and Pālitya

Doṣa	Pitta Pradhāna Tridoṣa
Dūṣya	Rasa, Rakta, Asthi and Sveda
Agni	Jaṭharāgni māndhya
Āma	Agni māndhya janya Āma
Srotas	Rasa, Rakta, Asthi and Sveda vaha
Srotoduṣṭi	Saṅga
Sañcāra sthāna	Rasāyanīṣ
Udbhava sthāna	Āmāśaya
Vyakta sthāna	Keśabhūmi
Adhiṣṭhāna	Keśabhūmi
Rogamārga	Bahya

Bheda and Lakṣaṇa of Khālitya

- ⊙ तेजोऽनिलाद्यैः सह केशभूमिं दग्ध्वाऽऽशु कुर्यात् खलतिं नरस्य।
  किञ्चित्तु दग्ध्वा पलितानि कुर्याद्धरिप्रभत्वं च शिरोरुहाणाम्॥ (Ca.Ci.26/132)

The Tejas (Dehoṣmā – body heat) associated with Vātādi doṣas quickly burn the Keśabhūmi (ground of scalp hair) and cause a person to suffer from Khālitya. If burning is less, it causes Palita (greyish) or Hariprabhatvaṃ (Kapilatva – *tawny or yellowish brown* colour) of Śiroruhāṇām (scalp hair).

- ⊙ रोमकूपानुगं पित्तं वातेन सह मूर्छितम् । प्रच्यावयति रोमाणि ततः श्लेष्मा सशोणितः ॥
  रुणद्धि रोमकूपांस्तु ततोऽन्येषामसम्भवः । तदिन्द्रलुप्तं खालित्यं रुह्येति च विभाव्यते ॥
  (M.N.55/28-29) / (Su.Ni.13/33-34)

Pitta combined with Vāta affects the Romakūpa *(hair follicles)* and does *Pracyāvayati* romāṇi (fall of hairs). Subsequently, Kapha mixed with Rakta does *Ruṇaddhi romakūpāstu* (blocks the hair follicles) and Tatoanyeṣāmasambhava *(prevents the growth of new hair)*. This condition is called as Indralupta, and it is also known as *Khālitya and Ruhyā*.

- ⊙ "इन्द्रलुप्तं श्मश्रुणि भवति, खालित्यं शिरस्येव, रुह्या च सर्वदेहे।" इति। (Ma.M.N.55/28-29)

Ācārya Mādhavakara, while commenting on the above verse, mentioned that the Indralupta occurs in Śmaśruṇi (beard area), *Khālitya occurs on the Śiras* (head), and Ruhyā occurs in Sarvadeha (all over the body)

Ācārya Vāgbhaṭa classified Khālitya *into four types, namely*, Vātaja, Pittaja, Kaphaja and Sannipātaja.

- ⊙ खलतेरपि जन्मैवं शातनं तत्र तु क्रमात्॥ (A.H.U.23/26)
- ⊙ खलतौ, क्रमेण केशानां शातो न त्विन्द्रलुप्त इव सहसा। (Aru. A.H.U.23/26-27)

Khālitya refers to the *Kramena keśāṇāṃ śāto* (the gradual process of hair loss). In contrast, Indralupta is characterized by *Sahasā keśāṇāṃ śāto* (sudden hair loss).

- ⊙ सा वातादग्निदग्धाभा, पित्तात्स्विन्नसिरावृता। कफाद्धनत्वग्वर्णाश्च यथास्वं निर्दिशेत् त्वचि॥
  दोषैः सर्वाकृतिः सर्वैरसाध्या सा नखप्रभा। दग्धाग्निनेव निर्लोमा सदाहा या च जायते॥ (A.H.U.23/26-28)
- ⊙ सा वातादग्निदग्धाभा पित्तात् स्वेदसिरावृता। कफात् घनत्वग्वर्णांश्च यथास्वं निर्दिशेत्त्वचि।
  दोषैः सर्वाकृतिः सर्वैरसाध्या सा नखप्रभा। दग्धाग्निनेव निर्लोमा सदाहा या च जायते॥ (A.S.U.27/21)

- रूक्षं सपाण्डुरं वातात्पित्ताद्रक्तं सदाहकम् । कफान्वितं भवेत् स्निग्धं रक्तात् पाकं व्रजन्ति तत् । सन्निपातेन सदृशं सर्वलक्षणम् ॥ (Hā.Sa.Tṛ.44/1-2)

  Ācārya Hārita classified Khālitya into five types by adding the Raktaja Khālitya.

  - **Vātaja Khālitya** causes Agnidagdhābhā (the scalp appears as if burnt by fire). Hārita added Rūkṣa (dryness) and Pāṇḍura (pale, whitish colour)
  - **Pittaja Khālitya** leads to Svinnasirāvṛtā (sweating and prominent veins seen in the scalp). Hārita added Raktaṃ sadāham (reddish colour with burning sensation)
  - **Kaphaja Khālitya** results in Ghanatvagvarṇā (thickening and discoloration of the skin of the scalp). Hārita added Snigdhaṃ (unctuous or oily)
  - **Raktaja Khālitya** leads to Pāka (suppuration)
  - **Sannipātaja Khālitya** is caused due to the involvement of all the doṣas, and it is considered as Asādhya (incurable). It resembles the Nakhaprabhā (nail in colour), looks as though Dagdhāgnineva (burnt by fire), Nirlomā (has no hairs) with Dāha (burning sensation).

Bheda and Lakṣaṇa of Pālitya

- तत् पलितं द्विविधं भवति, अकालजनितं कालजनितं च । (Āḍ.Śā.P.7/151-152)

  Palita is classified into two types namely

  - **Kālajanitaṃ** refers to greying of hair that occurs naturally with age
  - **Akālajanitaṃ** refers to premature greying of hair.

- क्रोधशोकश्रमकृतः शरीरोष्मा शिरोगतः । पित्तं च केशान् पचति पलितं तेन जायते ॥ (M.N.55/32)
- शोकश्रमक्रोधकृतः शरीरोष्मा शिरोगतः। केशान् सदोषः पचति पलितं सम्भवत्यतः॥ तद्वातात्स्फुटितं श्यावं खरं रूक्षं जलप्रभम्। पित्तात्सदाहं पीताभं, कफात् स्निग्धं विवृद्धिमत्॥ स्थूलं सुशुक्लं, सर्वैस्तु विद्याद्व्यामिश्रलक्षणम्। शिरोरुजोद्भवं चान्यद्द्विवर्णं स्पर्शनासहम्॥ असाध्या सन्निपातेन खलतिः पलितानि च। (A.H.U.23/29-32)
- शोकश्रमक्रोधकृतः शरीरोष्मा शिरोगतः। केशान् सदोषः पचति पलितं सम्भवत्यतः। तद्वातात्स्फुटितं श्यावं खरं रूक्षं जलत्प्रभम्। पित्तात्सदाहं पीताभं कफात्स्निग्धं विवृद्धिमत्। स्थूलं सुशुक्लं सर्वैस्तु विद्याद्व्यामिश्रलक्षणम्। शिरोरुजोद्भवं चान्यद्द्विवर्णं स्पर्शनासहम्॥ (A.S.U.27/22)

Due to Krodha (anger), Śrama (fatigue), and Śoka (grief), the Śarīroṣmā (heat of the body) enters the Śiras (head), getting mixed with the doṣas, resulting in Keśān pacati (ripens the hairs) and causes Palita (greying of hair). Pālitya is classified into five types

- **Vātaja Palita** leads to the hair becomes Sphuṭitaṃ (split), Śyāva (brown), Kharaṃ (rough), Rūkṣaṃ (dry) and Jalaprabham (watery hue)

- **Pittaja Palita** causes Dāha (burning sensation) and Pītābhaṃ (yellowish hair)
- **Kaphaja Palita** leads to Snigdhaṃ (unctuous), Sthūlaṃ (thick) and Suśuklaṃ (whitish hair)
- **Sannipātaja Palita** results in Miśra lakṣaṇās (mixed features)
- **Śirorujodbhavaṃ Palita** (greying of hair arises from headaches) causes Vivarṇa (discoloured) and Sparśanāsaham (intolerance to touch).

Sādhyāsādhyata of Khālitya and Pālitya

- असाध्या सन्निपातेन खलतिः पलितानि च। (A.S.U.27/23)

Khalati and Palita caused by Sannipāta (aggravation of all the doṣas simultaneously) is Asādhya (incurable).

In Contemporary Science,

Khālitya and Pālitya can be compared with Hair loss and premature greying of hair respectively in Contemporary Science.

MCQ's

1. Khālitya is

a) Nirlomaśirastvam
b) Śīrṇaromatva
c) Piñjaratva
d) All of the above

2. Pālitya is

a) Keśaśuklatva
b) Khalvāṭatvaṃ
c) Keśānāṃ patinaṃ
d) All of the above

3. Excessive intake of which rasa leads to Khālitya

a) Amla
b) Lavaṇa
c) Kaṭu
d) Kaṣāya

4. Khāltya is of .... types

a) 2
b) 3
c) 4
d) 5

5. Agnidagdhābhā is seen in

a) Vātaja Khālitya b) Pittaja Khālitya

c) Kaphaja Khālitya d) Tridoṣaja Khālitya

6. Nakhaprabhā is seen in

a) Vātaja Khālitya b) Pittaja Khālitya

c) Kaphaja Khālitya d) Tridoṣaja Khālitya

7. Palita is of .... types

a) 2 b) 3

c) 4 d) 5

Answer Keys

1	d	2	a	3	b	4	d	5	a	6	d	7	a

TOPIC 59

Ślīpada

Introduction

Ślīpada is a Kapha Pradhāna Tridoṣaja Vyādhi characterised by Kramena Pādagata Ṣotha (progressive swelling of the lower limb). Ācārya Caraka explained it under Śvayathu Cikitsā. Ācārya Vāgbhaṭa explained it under the Granthyarbudaślīpadāpacīnāḍīvijñānīya adhyāya. Ācārya Suśruta explain it under Vṛddhyupadaṃśaślīpadānāṃ Nidāna. Hastipāda, Goda and Pādavalbhīkam are the synonyms of Ślīpada.

Nirūkti and Paribhāṣa

◉ श्लीपदं स्फीतपादादि । (SKD)

That which causes Sphītapādādi (swelling of the feet and legs) is called Ślīpada

◉ शिलावत् पदं श्लीपदमिति । (Ma.M.N.39/1-3)

That which causes Śilāvat padaṃ (foot resembles stone) is called Ślīpada.

Nidāna and Saṃprāpti

There is no direct reference to the causes of Ślīpada in Samhitas. However, scattered references are available regarding the cause of Ślīpada. They are

◉ पुराणोदकभूयिष्ठाः सर्वर्तुषु च शीतलाः । ये देशास्तेषु जायन्ते श्लीपदानि विशेषतः ॥ (Su.Ni.12/14)/ (M.N.39/6)

Ślīpada occurs primarily in the Purāṇodaka bhūyiṣṭhā (the region with abundant ancient water) and Sarvarṭuṣu ca Śītalā (where water is cool in all seasons).

◉ पारियात्रभवा याश्च विन्ध्यसह्यभवाश्च याः। शिरोहृद्रोगकुष्ठानां ता हेतुः श्लीपदस्य च॥ (Ca.Sū.27/212)

The rivers originating from the Pāriyātra, Vindhya, and Sahya mountains cause Śirohṛdrogakuṣṭhānāṃ (diseases of the head and the heart and skin) as well as Ślīpada (elephantiasis).

- ⦿ तत्र नद्यः पश्चिमाभिमुखाः...... महेन्द्रप्रभवाः श्लीपदोदराणि, हिमवत्प्रभवा हृद्रोगश्वयथुशिरोरोगश्लीपदगलगण्डान् ॥ (Su.Sū.45/21)

Ācārya Suśruta, while explaining about the Nadī (river water), mentioned that the river originating from the Mahendra range yields Ślīpada (elephantiasis) and Udara (ascites). Furthermore, he observed that the river deriving from the Himavat range gives rise to Hṛdroga (heart disease), Śvayathu (swelling), Śiroroga (diseases of the head), Ślīpada (elephantiasis), and Galagaṇḍa (goiter).

- ⦿ यच्छलेष्मलाहारविहारजातं पुंसः प्रकृत्याऽपि कफात्मकस्य । सास्रावमत्युन्नतसर्वलिङ्गं सकण्डुरं श्लेष्मयुतं विवर्ज्यम् ॥ (M.N.39/7)

While explaining the unfavourable signs of Ślīpada, Ācārya Mādhavakara mentioned that Ślīpada is caused by the Śleṣmala āhara vihāra (food and lifestyle habits that increase Kapha) and particularly true even for individuals with a naturally Kapha Prakṛti. Therefore, Kaphaja Āhāra and Vihāra (consuming food and following a lifestyle that increases Kapha) can be considered causative factors of Ślīpada.

- ⦿ प्रस्थिता वङ्क्षणोर्वादिमधः कायं कफोल्बणाः॥ दोषा मांसास्रगाः पादौ कालेनाश्रित्य कुर्वते। शनैःशनैर्घनं शोफं श्लीपदं तत्प्रचक्षते॥ (A.S.U.34/18-19) / (A.H.U.29/18-19)

In the case of Ślīpada, doṣas, with the predominance of Kapha, invade the Māṃsa (muscle) and Rakta (blood) in the Adhaḥ Kāya (lower body). This invasion produces Śotha (swelling) initially in the Vaṅkṣaṇa (groin), followed by a gradual descent to the Pāda (feet), leading to a slow and progressive swelling.

- ⦿ जङ्घासु पिण्डीप्रपदोपरिष्टात् स्याच्छलीपदं मांसकफास्रदोषात्। (Ca.Ci.12/98)

Ślīpada manifests in the Jaṅghā (shank), Piṇḍīprapadopariṣṭāt (above the ankles or upper portion of the foot) due to the vitiation of Māṃsa, Kapha and Rakta.

- ⦿ कुपितास्तु दोषा वातपित्तश्लेष्माणोऽधःप्रपन्ना वङ्क्षणोरुजानुजङ्घास्ववतिष्ठमानाः कालान्तरेण पादमाश्रित्य शनैः शोफं जनयन्ति, तं श्लीपदमित्याचक्षते । (Su.Ni.12/10)

When the aggravated doṣas (Vāta, Pitta and Kapha) descend and reside in Vaṅkṣaṇa (groin), Ūru (thighs), Jānu (knees) and Jaṅghā (calves), it results in Kālāntareṇa Pādamāśritya śanaiḥ śophaṃ (gradual swelling in the feet). This condition is known as Ślīpada (elephantiasis).

- ⦿ त्रीण्यप्येतानि जानीयाच्छलीपदानि कफोच्छ्रयात् । गुरुत्वं च महत्त्वं च यस्मान्नास्ति कफं विना ॥ (Su.Ni.12/13)/ (M.N.39/5)

Ślīpada is mainly caused by Kapha due to its Gurutva (heaviness) and Mahatva (extensiveness), despite being influenced by three doṣas.

Saṃprāpti Ghaṭaka

Doṣa	Kapha Pradhāna Tridoṣa
Dūṣya	Tvak, Rakta, Māṃsa and Medas
Agni	Jaṭharāgni, Dhātvāgni māndhya
Āma	Jaṭharāgni Dhātvāgni māndhya janya
Srotas	Rasa, Rakta, Māṃsa and Medo vaha
Srotoduṣṭi	Saṅga
Adhiṣṭhāna	Vaṅkṣaṇa (groin), Ūru (thighs), Jānu (knees) and Jaṅghā (calves)
Udbhava sthāna	Āmapakvāśaya
Sañcāra sthāna	Sarva rasavāhini
Vyakta sthāna	Jaṅghā (shank), Piṇḍīprapadopariṣṭāt (above the ankles)
Rogamārga	Bāhya

Sāmānya Lakṣaṇās of Ślīpada

- यः सज्वरो वङ्क्षणजो भृशार्तिः शोथो नृणां पादगतः क्रमेण ।
  तच्छलीपदं स्यात् करकर्णनेत्रशिश्नौष्ठनासास्वपि केचिदाहुः ॥ (Su.Ni.12/15)
- पादवद्धस्तयोश्चापि श्लीपदं जायते नृणाम् । कर्णाक्षिनासिकौष्ठेषु केचिदिच्छन्ति तद्विदः ॥
- पाणिनासौष्ठकर्णेषु वदन्त्येके तु पादवत्। श्लीपदं जायते तच्च देशेऽनूपे भृशं भृशम्॥ (A.H.U.29/22)

Ślīpada is characterised by Vaṅkṣaṇajo bhṛśārtiḥ śotho nṛṇāṃ pādagataḥ krameṇa (swelling that originates in the groin area and gradually extends to the feet, accompanied by severe pain), and Jvara (fever). Additionally, some opines that it can also affects other parts of the body such as the Kara (hands), Karṇa (ears), Netra (eyes), Śiśna (penis), Oṣṭa (lips) and Nāsa (nose). Vāgbhaṭa added that this disease is very common in Ānūpa deśa (marshy region).

Bheda and Lakṣaṇās

- तत्त्रिविधं- वातपित्तकफनिमित्तमिति ॥ (Su.Ni.12/10)

Ślīpada is classified in to three types namely Vātaja, Pittaja and Kaphaja Ślīpada.

Vātaja Ślīpada

- तत्र, वातजं खरं कृष्णं परुषमनिमित्तानिलरुजं परिस्फुटति च बहुशः । (Su.Ni.12/11)
- वातजं कृष्णरूक्षं च स्फुटितं तीव्रवेदनम् । अनिमित्तरुजं तस्य बहुशो ज्वर एव च ॥ (M.N.39/1-3)
- परिपोटयुतं कृष्णमनिमित्तरुजं खरम्। रूक्षं च वातात् ...। (A.H.U.29/20)

Vātaja Ślīpada is characterized by Kṛṣṇa (black), Rūkṣa / Khara (rough), Paruṣa (hard), Sphuṭita / Parisphuṭati (cracked) of the affected skin, Tīvravedana animittaruja (severe pain without any specific cause), and frequently accompanied by Jvara (fever).

Pittaja Ślīpada

- पित्तजं तु पीतावभासमीषन्मृदु ज्वरदाहप्रायं च । (Su.Ni.12/11)
- पित्तजं पीतसङ्काशं दाहज्वरयुतं मृदु । (M.N.39/1-3)
- ....पित्तात्तु पीतं दाहज्वरान्वितम्॥ (A.H.U.29/20)

Pittaja Ślīpada is characterized by Pītasaṅkāśa/Pītāvabhāsa (yellowish appearance), Iṣanmṛdu (slightly soft) of the affected skin, and mostly associated with Dāha (burning sensation), and Jvara (fever).

Kaphaja Ślīpada

- श्लेष्मजं तु श्वेतं स्निग्धावभासं मन्दवेदनं भारिकं महाग्रन्थिकं कण्टकैरुपचितं च ॥ (Su.Ni.12/11)
- श्लैष्मिकं स्निग्धवर्णं च श्वेतं पाण्डु गुरु स्थिरम् ॥ (M.N.39/1-3)
- कफाद्गुरु स्निग्धमरुक् चितं मांसाङ्कुरैर्बृहत्। (A.H.U.29/20)

Kaphaja Ślīpada is characterized by Śveta varṇā (whiteness), Snigdhāvabhāsa (glossy appearance) of the affected skin associated with Mandavedana or Aruk (mild pain or painless), Bhārika (heaviness), Mahāgranthikaṃ (large glandular enlargements), and Kaṇṭakairupacitaṃ/Māṃsāṅkuraībṛhat (skin studded with thorns / studded with big sprouts of muscle fibres). The skin may also appear Pāṇḍu (paleness), Guru (heavy), and Sthira (hard).

Sādhyāsādhyata

- तत्र संवत्सरातीतमतिमहद्वल्मीकजातं प्रसृतमिति वर्जनीयानि ॥ (Su.Ni.12/12)
- वल्मीकमिव सञ्जातं कण्टकैरुपचीयते । अब्दात्मकं महत्तच्च वर्जनीयं विशेषतः ॥ (M.N.39/4)
- तत्त्यजेद्वत्सरातीतं सुमहत् सुपरिस्रुति॥ (A.H.U.29/21)

A physician should reject the Ślīpada if the swelling became Saṃvatsarātī / Abdātmakaṃ (one-year-old), Atimahadvalmīkajātaṃ (very large like ant-hill), Kaṇṭakaīrupacīyate (studded with thorns), and Prasṛtamiti (discharging).

◉ यच्छलेष्मलाहारविहारजातं पुंसः प्रकृत्याऽपि कफात्मकस्य । सास्रावमत्युन्नतसर्वलिङ्गं सकण्डुरं श्लेष्मयुतं विवर्ज्यम् ॥ (M.N.39/7)

That which arises from Śleṣmala āhara vihāra (food and lifestyle habits that increase Kapha), even for an individual of a Kapha Prakṛti, Sāsrāvam (with discharge), Atyunnatasarvaliṅga (excessively elevated all features) and Kaṇḍu (itching sensation) associated with Kapha, should be rejected for the treatment.

In Contemporary Science,

Ślīpada is often correlated with Elephantiasis or Filariasis in contemporary science. Elephantiasis is a condition characterized by severe swelling, typically in the legs, caused by parasitic infections such as those from the filarial worms. These worms block the lymphatic system, leading to the characteristic swelling and thickening of the skin and tissues.

MCQ's

1. Ślīpada synonyms
   a) Hastipāda b) Goda
   c) Pādavalbhīkam d) All of the above
2. Doṣa Predominance in Ślīpada
   a) Vāta b) Pitta
   c) Kapha d) Sannipātaja
3. Ślīpada causes
   a) Śilāvat padaṃ b) Sphītapādādi
   c) Pādagata Śotha d) All of the above
4. According to Caraka, rivers which origin causes Ślīpada
   a) Pāriyātra b) Vindhya
   c) Sahya d) All of the above
5. Ślīpada is of .... types
   a) 3 b) 4
   c) 5 d) 7
6. In Slīpada, Śotha begins from
   a) Pāda b) Vaṃkṣaṇa
   c) Udara d) Uru

Answer Keys

1	d	2	c	3	d	4	d	5	a	6	b

TOPIC 60

Tuberculosis

Introduction

Tuberculosis (TB) is a chronic and progressive infectious disease caused by Mycobacterium tuberculosis. It primarily impacts the pulmonary system, it can also manifest in extrapulmonary sites, including the kidneys, spine, and brain. The global prevalence of active Tuberculosis in 2022 was estimated at 10.6 million individuals, making it the second leading cause of mortality with 1.3 million deaths, after COVID-19. The World Health Organization (WHO) has identified India as the country with the highest burden of Tuberculosis, with an estimated incidence of 210 cases per 100,000 individuals. Furthermore, the prevalence of Tuberculosis was observed to be higher among males compared to females.

Etiopathogenesis

Mycobacterium tuberculosis stands as a primary causative agent of this disease. Occasionally, similar illnesses can be caused by closely related mycobacteria such as M. bovis, M. africanum, M. canettii, and M. microtia. These mycobacteria, in conjunction with M. tuberculosis and other infrequent mycobacteria collectively constitute the Mycobacterium tuberculosis complex.

Mycobacterium tuberculosis is disseminated through aerosolized particles referred to as droplet nuclei, which typically measure 1-5 microns in diameter and encapsulate tubercle bacilli. These particles are predominantly propelled through activities such as coughing, singing, and other deliberate respiratory maneuvers by individuals afflicted with active pulmonary or laryngeal TB. It is noteworthy that the sputum of these individuals harbors a substantial quantity of organisms, approximately 10,000 organisms per milliliter, constituting the threshold of detection attainable through fluorescent microscopy.

Tuberculosis manifests in three distinct stages: Primary infection, Latent infection, and Active infection. The initial phase involves M. tuberculosis bacilli inducing a primary infection, with a small percentage progressing to varying degrees of clinical disease. Notably, approximately 95% of primary infections are asymptomatic. Following the initial infection, an indeterminate percentage may spontaneously resolve, whereas the majority enter a latent (dormant) phase. Subsequently, a variable percentage (5 to 10%) of latent infections may reactivate, exhibiting symptoms and signs of disease.

Primary tuberculosis infection commences when mycobacteria infiltrate the alveolar air sacs of the lungs and subsequently replicate within endosomes of alveolar macrophages. These macrophages recognize the bacterium as foreign and endeavor to eliminate it through phagocytosis. Consequently, the bacterium becomes encapsulated by the macrophage and transiently stored within a membrane-bound vesicle termed a phagosome. Subsequently, the phagosome merges with a lysosome to generate a phagolysosome. Within the phagolysosome, the cell employs reactive oxygen species and acid to eradicate the bacterium; however, the thick waxy mycolic acid capsule of M. tuberculosis confers protection against these harmful substances. M. tuberculosis can increase within the macrophage and ultimately induce the demise of the immune cell.

Latent tuberculosis infection typically follows a primary infection. In about 95% of cases, the immune system suppresses bacillary growth before symptoms appear. Tubercle bacilli can survive for years, and the infection outcome depends on the host's resistance and microbial virulence. Infectious foci may cause fibronodular scarring in the lungs (Simon foci) or small consolidations (Ghon foci). A Ghon focus accompanied by lymph node involvement is characterized as a Ghon complex, which, if calcified, is denoted as a Ranke complex. Both the tuberculin skin test and interferon-gamma release blood assays (IGRA) yield positive results during the latent stage of infection. In some cases, the primary focus may progress rapidly, causing acute illness with pneumonia, pleural effusion, and lymph node enlargement.

Active tuberculosis infection may develop in healthy people who are infected with tuberculosis and have a 5 to 10% lifetime risk, with the percentage varying significantly based on age and additional risk factors. Reactivation of the infection can occur within the first two years in 50 to 80% of cases, or it may manifest decades later, with the lungs being the most common site for the infection.

Risk factors

Individuals with an elevated risk of tuberculosis progression comprise those with HIV infection, undergoing immunosuppressive therapy, afflicted with conditions such as silicosis, diabetes mellitus, chronic renal failure, leukemia, or cancer. Additional risk factors encompass malnutrition, poverty, smoking, alcohol or substance abuse, and children under five years of age. Furthermore, healthcare workers and incarcerated populations are also at an increased risk.

Clinical features

Pulmonary tuberculosis is traditionally classified as primary or post-primary tuberculosis. However, molecular evidence from tuberculosis endemic areas challenges this categorization by indicating that a substantial proportion of adult pulmonary tuberculosis cases arise from recent infection (primary infection or reinfection) rather than from reactivation.

- **Primary Pulmonary tuberculosis** typically manifests soon after the initial infection with tubercle bacilli, often afflicting children or young adults. It usually presents as asymptomatic or with mild symptoms, such as low-grade fever, fatigue, weight loss, and a persistent cough. In children, lethargy, vague ill-health, failure to thrive, and developmental delays may be initial indicators. Strong clinical suspicion is crucial for diagnosing the disease at this stage. Additionally, allergic manifestations like phlyctenular conjunctivitis (1–3mm shiny yellow or grey bleb at limbus) or erythema nodosum (bluish red, raised, tender lesions over shin and thigh), may be early signs in some cases. Furthermore, primary tuberculosis can predispose individuals to recurrent respiratory infections, and acute pleural effusion may develop in some cases. Other presentations in children include tuberculous pneumonia, hemoptysis, or asthma. Physical examination may not yield positive findings in many patients, necessitating diagnostic reliance on investigations. However, in some cases, signs of pulmonary consolidation, collapse, or pleural effusion may be present. A positive tuberculin skin test is indicative in the majority of cases, and blood examination often shows elevated erythrocyte sedimentation rate (ESR) and lymphocytosis. Radiographic evidence of hilar lymphadenopathy, with careful examination, may also reveal detectable pulmonary lesions.
- **Post-primary pulmonary tuberculosis,** also known as adult post-primary pulmonary tuberculosis, can occur from the reactivation of latent TB infection or recent infection (primary infection or reinfection). The disease develops gradually and presents symptoms such as fever, night sweats, malaise, loss of appetite, weight loss, and cough, often accompanied by haemoptysis. Initially, there may be little sputum, which later becomes more copious and of a mucoid or mucopurulent nature. Mild streaky haemoptysis may progress to severe haemoptysis due to erosion of hypertrophied bronchial vessels (Rasmussen's aneurysm). In some cases, the initial presentation might involve pleural effusion, empyema, or pneumothorax. As the disease progresses, the patient experiences extreme weight loss, pronounced cough with expectoration, and the development of advanced Cachexia (phthisis). Upon physical examination of the chest, signs of consolidation, cavitation, collapse, fibrosis, pleural effusion, or pneumothorax may be revealed.

Extrapulmonary tuberculosis predominantly affects sites such as the lymph nodes, pleura, genitourinary tract, bones and joints, meninges, peritoneum, and pericardium, in descending order of prevalence. Nonetheless, it can impact nearly all organic systems. Owing to hematogenous dissemination in individuals afflicted with HIV, extrapulmonary tuberculosis is presently more prevalent in regions characterized by a high incidence of HIV infection than in the past.

- **Lymph Node TB (Tuberculous Lymphadenitis)** is a form of tuberculosis that affects the lymph nodes. It is one of the most common presentations of extrapulmonary tuberculosis. Lymph node TB presents as painless swelling of the lymph nodes, often in the cervical (neck) region. Other symptoms can include fever, night sweats, weight loss, and fatigue. The diagnosis is established by fine-needle aspiration biopsy or surgical excision biopsy.

- **Pleural tuberculosis (Pleural TB)** is a manifestation of extrapulmonary tuberculosis affecting the pleura, the thin membrane encompassing the lungs. Pleural TB can occur when tuberculosis from the lung spreads to the pleura. Depending on the severity, the effusion may be minor and resolve on its own, or it may be substantial enough to cause symptoms such as fever, pleuritic chest pain, cough, and dyspnoea. Diagnosing pleural TB typically involves imaging studies like chest X-rays or CT scans, as well as microbiological tests such as pleural fluid analysis, pleural biopsy, and culture.

- **Genito-urinary tract Tuberculosis (GUTB)** denotes an extrapulmonary form of tuberculosis affecting the urinary system, genital organs, or both. Its clinical presentation encompasses increased urinary frequency, dysuria, nocturia, haematuria, and flank or abdominal pain. In males, testicular and epididymal involvement may induce sterility, while in females, fallopian tube impairment can lead to infertility. The diagnostic protocol for GUTB typically entails early morning urine cultures, intravenous pyelogram (IVP), and occasionally cystoscopy.

- **Bone and Joint Tuberculosis (Skeletal Tuberculosis)** is affects weight bearing joints especially spines (Pott's disease or tuberculous spondylitis), hips and knees. It presents with pain, joint swelling and paraosseous 'cold' abscesses (because they lack the typical signs of inflammation like warmth and redness) and sinus tract formation. Diagnosis requires aspiration and biopsy of the synovial fluid.

- **Tuberculous Meningitis (TBM)** represents a severe and potentially life-threatening manifestation of meningitis, often arising from the acute dissemination of primary or postprimary pulmonary TB through the hematogenous route. Alternatively, TBM may occur due to the rupture of a subependymal nodule into the subarachnoid space. Clinical presentation typically comprises subtle symptoms including persistent headaches, fever, neck stiffness, altered mental status, and neurological deficits, such as cranial nerve palsies. In severe cases, the condition can progress to coma and fatality. Diagnosis necessitates the analysis of cerebrospinal fluid (CSF) obtained through lumbar puncture.

- **Gastrointestinal Tuberculosis** is a relatively infrequent occurrence, often attributed to the ingestion of sputum containing tubercle bacilli, hematogenous dissemination, or consumption of milk from cows affected by bovine tuberculosis. The terminal ileum and the caecum are the most commonly affected areas. Clinical manifestations encompass abdominal discomfort, unexplained weight loss, pyrexia, alterations in bowel habits, nausea, vomiting, and hematochezia. Non-specific abdominal pain, fever, and ascites should prompt consideration of tuberculous peritonitis. Definitive diagnosis requires histological assessment and culture of specimens obtained intraoperatively.

- **Pericardial TB (Tuberculous Pericarditis)** represents a consequential manifestation of extrapulmonary tuberculosis that specifically impacts the pericardium. This condition may arise through direct extension from nearby mediastinal or hilar lymph nodes, or via hematogenous dissemination from a primary infection site. Clinical manifestations of pericardial TB are often gradual and nonspecific, encompassing fever, fatigue, night sweats, weight loss, chest discomfort, dyspnea, and cough. In severe instances, potential complications may include cardiac tamponade and constrictive pericarditis. Definitive diagnosis entails the detection of tubercle bacilli in pericardial fluid or histological evidence of caseating granulomas.

Miliary tuberculosis (Miliary TB) is a form of tuberculosis that affects both the pulmonary and extrapulmonary systems. It commonly manifests in young children and adults and is attributable to the acute dissemination of TB bacilli via the bloodstream to multiple organs, leading to the emergence of discrete nodular shadows approximately the size of a millet seed (2 mm). Symptoms of miliary TB include significant pyrexia, drenching night sweats, tachycardia, weight loss, and a dry cough. Hepatosplenomegaly may be present, and the presence of headaches may indicate a concurrent diagnosis of tuberculous meningitis. Fundoscopy may reveal the existence of choroidal tubercles. Radiographic examination typically reveals the presence of fine 1–2-mm lesions ('millet seeds') distributed throughout the pulmonary system. Ultrasonography, contrast-enhanced computed tomography (CECT), and magnetic resonance imaging (MRI) are valuable modalities for assessing extrapulmonary TB lesions, offering insight into the extent of organ involvement. In recent practice, positron emission tomography computed tomography (PET-CT) has been utilized as a means of investigating patients suspected of TB.

Diagnosis: Diagnosis is based on the clinical evaluation, Imaging studies, Acid fast bacilli (AFB) test, tuberculin skin test and biopsy. Clinically, Pulmonary TB is typically suspected in patients presenting with a cough persisting for two weeks or more, chest pain, unexplained fever, or haemoptysis.

Imaging studies particularly chest X-rays, can reveal ill-defined opacities in the apex, initially appearing unilaterally and later indicating bilateral involvement. These studies may also provide evidence of fibrosis, cavities, consolidation, pneumothorax, or pleural effusion. Additionally, CT and MRI imaging techniques can aid in assessing the extent of TB in the lungs and other organs. Positron Emission Tomography-Computed Tomography (PET-CT) is also utilized to evaluate the extent of TB involvement in various organs.

The presence of Acid Fast Bacilli (AFB) can be identified in sputum smear and sputum culture using the Ziehl-Neelsen technique. Nucleic Acid Amplification Tests (NAATs) can rapidly detect TB bacteria and ascertain their drug resistance.

The Tuberculin skin test (Mantoux test) entails injecting one unit of purified protein derivative (PPD) intradermally into the volar surface of the forearm. Following 48-72 hours, the injection site is examined for swelling. A positive reaction would indicate TB infection.

Tissue biopsy from affected areas, such as lymph nodes or the pleura, can be examined for TB bacteria.

MCQ's

1. Which bacterium is responsible for causing tuberculosis?

a) Staphylococcus aureus
b) Escherichia coli
c) Mycobacterium tuberculosis
d) Pseudomonas aeruginosa

2. What is the most common site of extrapulmonary tuberculosis?

a) Liver
b) Lymph nodes
c) Kidneys
d) Brain

3. Which diagnostic test involves injecting a small amount of tuberculin into the skin?

a) Chest X-ray
b) Sputum culture
c) Tuberculin Skin Test (TST)
d) Interferon-Gamma Release Assay (IGRA)

4. What is the characteristic radiographic finding in miliary tuberculosis?

a) Large cavitary lesions
b) Pleural effusion
c) Fine 1–2-mm nodular lesions
d) Consolidation in the lower lobes

5. Which of the following is not a common symptom of tuberculous meningitis?

a) Persistent headache
b) Neck stiffness
c) High blood pressure
d) Altered mental status

6. What is the standard duration of treatment for most forms of tuberculosis?

a) 2 weeks
b) 1 month
c) 3 months
d) 6 months or more

7. Which imaging technique is often used to assess the extent of organ involvement in extrapulmonary TB?

   a) Ultrasound
   b) CT Scan
   c) MRI
   d) All of the above

8. Which of the following is a common complication of tuberculous pericarditis?

   a) Cardiac tamponade
   b) Pulmonary embolism
   c) Myocardial infarction
   d) Aortic dissection

9. Which diagnostic method is used to confirm the presence of tuberculosis bacteria in the pericardial fluid?

   a) Chest X-ray
   b) Sputum culture
   c) Histological examination
   d) Tuberculin Skin Test (TST)

10. Which of the following statements about primary pulmonary tuberculosis is true?

    a) It typically affects older adults.
    b) It usually presents with severe symptoms
    c) It often presents as asymptomatic or with mild symptoms.
    d) It is always associated with haemoptysis.

11. What is a common allergic manifestation of primary pulmonary tuberculosis in children?

    a) Phlyctenular conjunctivitis
    b) Erythema nodosum
    c) Both A and B
    d) Neither A nor B

12. What is a potential complication of post-primary pulmonary tuberculosis due to erosion of hypertrophied bronchial vessels?

    a) Pleural effusion
    b) Severe haemoptysis
    c) Pneumothorax
    d) Empyema

13. Which of the following symptoms is NOT commonly associated with pleural tuberculosis (Pleural TB)?

    a) Pleuritic chest pain
    b) Cough
    c) Dyspnoea
    d) Haematuria

14. Which joints are most commonly affected by bone and joint tuberculosis (Skeletal Tuberculosis)?

a) Elbows and wrists
b) Spines, hips, and knees
c) Shoulders and ankles
d) Fingers and toes

15. Which part of the gastrointestinal tract is most commonly affected by gastrointestinal tuberculosis?

a) Stomach
b) Terminal ileum and caecum
c) Oesophagus
d) Rectum

16. What is the primary method for diagnosing tuberculous meningitis (TBM)?

a) Chest X-ray
b) Sputum culture
c) Lumbar puncture and cerebrospinal fluid (CSF) analysis
d) Blood culture

Answer Keys

1	c	2	b	3	c	4	c	5	c	6	d	7	d	8	a
9	c	10	c	11	c	12	b	13	d	14	b	15	b	16	c

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